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# *EPOCH-MAKING BOOKS IN BRITISH SURGERY.*

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## I. "A SYSTEM OF SURGERY" BY MASTER JOHN ARDERNE.

A FEW books remain outstanding from each of the great periods of British surgery. Arderne's *System of Surgery*, the result of the Hundred Year's War with France; the writings of Gale, Clowes, and Read in the struggle for the recognition of surgery as a profession during the Tudor period; the books of Peter Lowe, Woodall, and Wiseman under the Stuarts; the treatises of Percivall Pott, John Hunter, and Sir Astley Cooper under the Hanoverians; and lastly, of Lord Lister in our own days. All are worthy to be read and re-read, for all exercised a marked influence upon their contemporaries, and through them upon ourselves. It is the purpose of this series of short articles to examine the writings and to show the means by which they attained their importance.

I have written at some length on former occasions about Master John Arderne (1307–1380 ?) as the first great operating surgeon in England whose writings are still extant. There is little to be said about him personally that is new, except that, browsing in the Record Office last year, I found two documents dealing with his domestic affairs. The first is dated 1348, when he was 41 years of age. By it he agrees, jointly with Ellen his wife, to pay six shillings and eightpence for 18 acres of land, and 1½ acres of meadowland at Mitcham in Surrey, together with two shillings and sixpence rent. Three years later, in 1351, he was granted a licence to transfer the whole of the land to John de Ware, but there is no mention of Ellen his wife. The story is a sad one, as I read it. He came home from the wars after the battle of Creçy in 1346, a successful surgeon with many influential friends. He married and settled with his wife at Mitcham. Within a year his wife died, perhaps in childbed, more likely of the Black Death, for he says himself that "he dwelled in Newark in Nottinghamshire from the first pestilence that was in the year of our Lord 1349, unto the year of our Lord 1370". And these two dates, with that of his birth and of the death of the Black Prince, are all that

he cares to remember. It is evident that he sold his newly-bought estate and moved to Newark, where he would be welcomed by the Earl of Lancaster, under whom he had served, and later by John of Gaunt. He built up a large surgical practice amongst the great landowners, and in his old age he set himself to write a *System of Surgery*.

The *System* consists of a series of treatises which could be bought separately. Manuscripts were expensive at the time they were written, and surgeons would buy only the section dealing with the subject in which they were interested. Some of the sections, therefore, had a large circulation, others had a smaller sale, if we may judge by the number of manuscripts of the Treatise on Fistulæ, as compared with those on the Diseases of Women, which are still to be found in our public libraries. Originally written in Latin " propria manu ", with my own hand, as he says in the treatise on Diseases of the Eye, they were soon translated into English, some by North countrymen, others by scribes of the South Country. They were largely read, but they were not reproduced during the troubled times of the Wars of the Roses, and, having escaped the printing press at the end of the fifteenth century, there was some danger of their being entirely forgotten. Fortunately the treatises were collected into a portly volume entitled *Practica Magistri Johannes de Arderne*. One is in Emmanuel College, Cambridge, and another is in the Library of the British Museum. The latter, from which the Keeper of the Manuscripts has allowed me to reproduce *Plates I* and *II*, is beautifully illustrated and may have been a presentation copy. The work of collecting the treatises has not been done skilfully in either case. No method has been adopted, and parts of one section have been allowed to intervene, so that it is only by reading the whole that the work of Arderne can be properly understood. When this is done it is obvious that Arderne's collected works form a fairly complete system of surgery as it was then understood, when surgery had not yet been divorced from medicine and when neither medicine nor surgery had freed itself from the herb lore of Saxon times, even for so advanced a surgeon as John Arderne.

The first section of the *System* deals with Hæmorrhage. It states when a patient should be bled, and gives formulæ for stopping bleeding in cancer, in bleeding piles, and in menorrhagia. Another section treats of Diseases of the Eyes, another of Diseases of the Male Generative Organs, and another of Diseases of Women. There is an excellent section on Diseases of the Kidney, with advice to those who suffer from renal calculus. The symptoms and treatment of Intestinal Obstruction are described with admirable vividness, and Gout is treated of at length, for it was a common complaint amongst the wealthy nobles who lived luxuriously at a time when there were no satisfactory purgatives. Fistulæ, whether from festering wounds or of the ordinary rectal type, are also dealt with exhaustively. They were common as a result of arrow wounds and when knights in heavy armour spent long days in the saddle in all weathers. Incidentally there is a supplementary section into which Arderne threw a whole series of interesting cases which had come under his observation at different times.

*Cancer of the Male Breast.*—The great merit of the *System* is that the author writes from a clinical standpoint. The general scheme of each section

is to illustrate the diseases from personal experience and to give details of the patients whom the author himself had treated. It is still readable, therefore, for it gives a picture not only of the practice and trials of a surgeon but also of the conditions under which a patient lived in the fourteenth century. Here is a good example of his method of writing. The case was clearly a cancer of the male breast: "To a priest of Colstone near Bynghame [Colston Bassett in Nottinghamshire] there fell a sore in the right pap within the skin, upon the top of the pap as it were a little knot about the size of a pea with itching. And the aforesaid knot grew continuously till it was as large as the egg of a hen and looked like a top. The colour of the sore was livid, mingled with red, and it discharged a wateriness. It felt hard, and when two years were passed he was taught by a lady to lay a plaster upon it and to drink the drink of Antioch for a long time. And when he perceived that the aforesaid medicines availed him nothing he went into the town of Nottingham to be let blood, and when the barber perceived the aforesaid knot he asked of him whether he would be helped about it and told him that he knew of a cure for it and could heal him. The priest said he would fain be helped, but nevertheless he said to him he would ask counsel if it might be done to him as he said. And in the same town there was a wise surgeon [i.e., John Arderne himself] of the which the said priest had knowledge, and he went to him and asked counsel if that he were curable, or if that he might suffer any cutting or corrosive or any such other medicines. And the said leech warned him that he should on no manner put no corrosive nor any other violent medicines nor allow any cutting to be done, for if he did he promised that it would bring him to his death without any recovery".

*A Case of Traumatic Tetanus.*—There was "a gardener who, while he worked amongst the vines, cut his hand with a hook upon a Friday after the feast of [the translation of] Saint Thomas of Canterbury in summer [July 7], so that the thumb was wholly separated from the hand except at the joint where it was joined to the hand, and it could be bent backward to his arm and there streamed out much blood.

"And as touching the cure. The thumb was first reduced to its proper position and sewn on and the bleeding was stopped with Lanfrank's red powder and with the hairs of a hare and the dressing was not removed until the third day. When it was removed there was no bleeding. Then there was put upon it those medicines which engender blood, redressing the wound once every day. The wound began to purge itself and to pour out matter. And on the fourth night after, the blood began to break out about midnight and he lost almost two pounds of it by weight. And when the bleeding was stopped the wound was re-dressed daily as before. Also on the eleventh night about the same time the bleeding broke out again in greater quantity than it did the first time. Nevertheless the blood was staunched, and by the morning the patient was so taken with the cramp in the cheeks and in the arm that he was not able to take any meat into his mouth, nor could he open his mouth, and on the fifteenth day the bleeding broke out again, and on the eighteenth day the blood broke out again beyond all measure, and always the cramp continued and he died on the twentieth day".

He says, describing a case of facial paralysis, that " it is a kind of cramp which turneth the mouth of a man or woman down to the ear in the manner of a fish called a flounder. Note well, if the crookedness of the mouth endure for six months it shall never be cured ". Incidentally he seems to have seen several cases of diphtheria, for he begs his readers to " note this diligently that in quinsy and in all swellings of the throat and neck and in all difficulty of swallowing, as when the patient may not be able to swallow meat or drink, I have seen many die thereof within five days through strangling ".

There is a clear description of intestinal obstruction under the name of passio iliaca. " It cometh with constipation of the belly and busy casting [vomiting] and with huge acheing as though the guts were being bored with a wymball [gimlet]. And it hath this difference from Colic, for in Iliac there is more ache and more wallowing now on the right side and now on the left side, and it sooner slayeth through its torment than doth the Colic. And in the Iliac the ache is most from the navel upwards, and the matter that should pass out beneath-forth cometh out of the mouth and in other cases there happeneth many cursed sicknesses as hiccoughing, the cramp, and coldness and stinking of all the body. And all these be mortal signs if they be with continual vomiting ". As regards colic it is possible to distinguish renal colic from colic of the bowels because in renal colic " the sorrow of the reynes is felt whilst going to and fro ", i.e., when walking or riding.

Arderne distinguishes between inflammation of the kidney and a botch or abscess of the kidney, and between a botch of the kidney and a botch of the bladder. Botch of the bladder leads him to speak of strangury, and strangury of the chaudepisse or " burning of the urine ", i.e., gonorrhœa, with which he was familiar and treated with mucilaginous injections. He also gives details of a case of soft sores which had been unsatisfactorily treated by " a lady " before the patient applied to Arderne.

Dysuria is classified into the cases " when the urine is utterly denied ", i.e., suppression, which " is a sign of death within seven days ", and " difficulty of pissing " or retention.

A stone impacted in the urethra " causeth the patient to suffer right great pain and intolerable sorrow. It may be pushed back into the bladder by an instrument of silver or latten such as may be made in every good town by Craftsmen that maketh pins for women's heads or at the goldsmiths. Many a one have I so holpen. I have seen young men and old in the which the stones have been so great that they could neither come out by the eye of the yard nor be pushed back but always abideth in the middle of the yard. And I cured them easily and quickly in this manner : First, I took the patient and laid him grovelling and then I bound the yard on both sides of the stone with linen bands, so that the stone might nowhere flee away, and with a little incision upon the stone with a lancet or with a razor I had out the stone, and after that I sewed the outer skin over the hole with a needle and thread, and then I laid on a dressing of white of an egg mixed with finely ground wheaten flour and left it for three days, and within fifteen days I had him perfectly cured ".

There is an interesting sidelight upon the chivalrous period in which the *System* was written. A modern writer identifies his patients by giving their

initials; Arderne does so by tricking their armorial bearings. He gives the following account of a patient: "I saw a man who could not move nor lift either foot or leg, nor could he draw up his foot, and even though it were put to a hot fire it would have been burnt, for he felt nothing, and yet he suffered no great pain and he was well enough in every other part of his body and could eat, drink and sleep well, but he could not go to stool on account of the constipation of his belly". The shield (*Plate II*) put against this passage was used by William, Lord of Douglas, who is said to have died of fever after a short illness in 1384. In the light of this record it seems that he had been under treatment for a slight paralytic attack, so that his fatal illness may have been due to a cerebral hæmorrhage. This William, as the first Earl Douglas, was surety for the ransome of David II, and was frequently in England. In November, 1363, King Edward III gave him a gilt cup of the value of £10 : 18 : 0. In like manner we learn that Lord de Grey, who served in Gascony in 1366, had a necrosis of his tibia; that Framlingham of Suffolk had tenesmus; that Crioll of Leicester had "a flux of the belly"; that Darbleton of Buck in Lincolnshire had an inflammation of the knee, possibly a staphylococcic infection starting in his tonsils, for it began with headache and a pain in his throat. There are also other shields belonging to the knightly families of the period, and all are still traceable by those skilled in heraldry.

Sometimes, as in *Plate I*, the illustrations are simply added for the sake of embellishing the manuscript. The king here depicted was probably intended for Demetrius III, King of Georgia, who died in 1289. Arderne may have heard the story from Sir Geoffrey Scrope, who was killed in Lithuania in 1362, the eldest son of Sir Henry le Scrope of Masham in Yorkshire. It runs, according to the text: "With this medicine [Gum Arabic dissolved in ink and used as an enema] was cured Demetrius, King of Perses, by a Christian man that was prisoner with the same king; which Christian man was made full rich and was sent home to Christian men's lands by commandment of the same king, and many prisoners of Christian men were let go free with him." The plant is "alleluya, i.e., wood sour, a trefoil growing under shrubs and bearing white flowers. Bruised and put upon hæmorrhoids, or condylomata or dead flesh it freteth softly and removeth all the above said things better than any thing in the world". Perhaps the helmet surmounted by a rat was the cognizance of Geoffrey Scrope.

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The Plates shown on pages 7 and 9 are copied by the kind permission of the Keeper of the Manuscripts at the British Museum.

The following transcription of the two pages of illustration is supplied for those who are unskilled in reading fourteenth century manuscripts:—

*Plate I.*

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ita ut per nastare possit in ano infundi. Cum hac medicina curatus fuit Demetrius rex persarum a quodam Christiano in captivitate apud dictum regem retento. Qui quidam Christianus ditissimus factus est et ad terram Christianorum iussu dicti regis transmissus et cum eo multi captivi de Christianis cum ipso liberi dimissi sunt.

Nota lucide alliaria.

Panis cuculi, anglice Wodesoure, et crescit ob dumis, id est busches, et est quoddam trifolium sicut hic depingitur et fert flores albos et est herba ualde acida et eadem dicitur alliaria panis igitur cuculi. Manipulum minimum in drapella lineo in aqua madefacto et expresso sine contricione uoluatur uel in folijs lappacij acuti, id est reddok, et sub cineribus calidis coquatur ut non desiccetur postea extrahatur et conteratur. Ponatur super emoroidas uel ficus uel condilomata uel carnem mortuam ubicunque fuerit et ficus corrodit suauiter et remouet omnia predicta super omni re de mundo. Quando ergo commode potest haberi temptare non nocet.

Nota de experimento regali.

Experimentum regale quod errorem tam prime digestiue quam jucunde feliciter ac mirifice emendat colorem que scelestum et innaturalem deponit et naturalem restaurat et bonum colorem facit. Recipe cynamoni zinziberis, galange, reubarbi antea subtilissime pulverizatur. Quibus addatur zucari equaliter et inuicem. Tere. Hoc puluere utatur paciens in principio prandij ad pondus quinque denariorum in vino.

Ista etiam medicina utilis est emoroidarum pacientibus quia frequenter sunt discolorati.

Nota de sputo sanguinis.

Contra epinia, id est sputum saniosum, et ulceracionem pulmonis et spiritualium. Recipe radices simphici, id est comferye, radices liquirici et succum eiusdem et semen Nasturcij, id est cresses, et semen plantaginis antea tere et cum melle confice et fiat electuarium ad predicta.

Nota quod succus liquirici est quedam confeccio facta de succo liquirici in transmarinis partibus et fit in latis laminibus ad modum glutis et est similis gluti et apotecarij hoc habent ad vendendum et vocatur Iude licorys id est Iuys licorys.

Nota de emoroidarum dolentibus.

Emoroidarum dolencium et inflatarum sine fluxu sanguinis fiat talis cura. Et est sciendum quod si bono modo possent fieri non deberent localia remedia mitigatiua apponi nisi prius corpus mundificaretur cum farmacia, id est medicina laxitiua, ut est de mirobolanis predictis et flebotomia quia aliter mitigatiua minus ualerent.

Mitigatiua pro emoroidis.

Mitigatur dolendum ad emoroidas inflatas cum punctura et arsura ex calida causa ut ex ignita uel adusta. Est emplastrum de folijs malue communis in aqua decoctis et postea cum axungia porci bene contritis addito oleo rose, uel uiole, uel lilij, uel camomille, uel communi si predicta non habeantur. Quo facto folia malue aqua expressa cum aliquo predictorum oleorum in patella bene frigentur: Postea super lanam carpinatam, uel stupas lini subtiles, de predictis maluis appone et calide applica miro modo mitigat dolorem. Si uero radix lilij poterit haberi et cum maluis coqueretur, ut dictum est, efficacius erit; et hoc emplastrum est satis commune quia in hyeme possunt haberi radices lilij potes facere ut de maluis dictum est, et si ambo haberi potuerit, efficacius erit emplastrum.

Item pro eodem.

Item mitigetur dolor et inflacio emoroidarum cum uitello oui dure assato et addito oleo rose et croco quibus inuicem contritos et cum lana apposita miro modo mitigat in omni causa et melius si predictis adderetur opium. Quorum si opium imponatur hec est proportio. [The quantity is not given in the MS.]

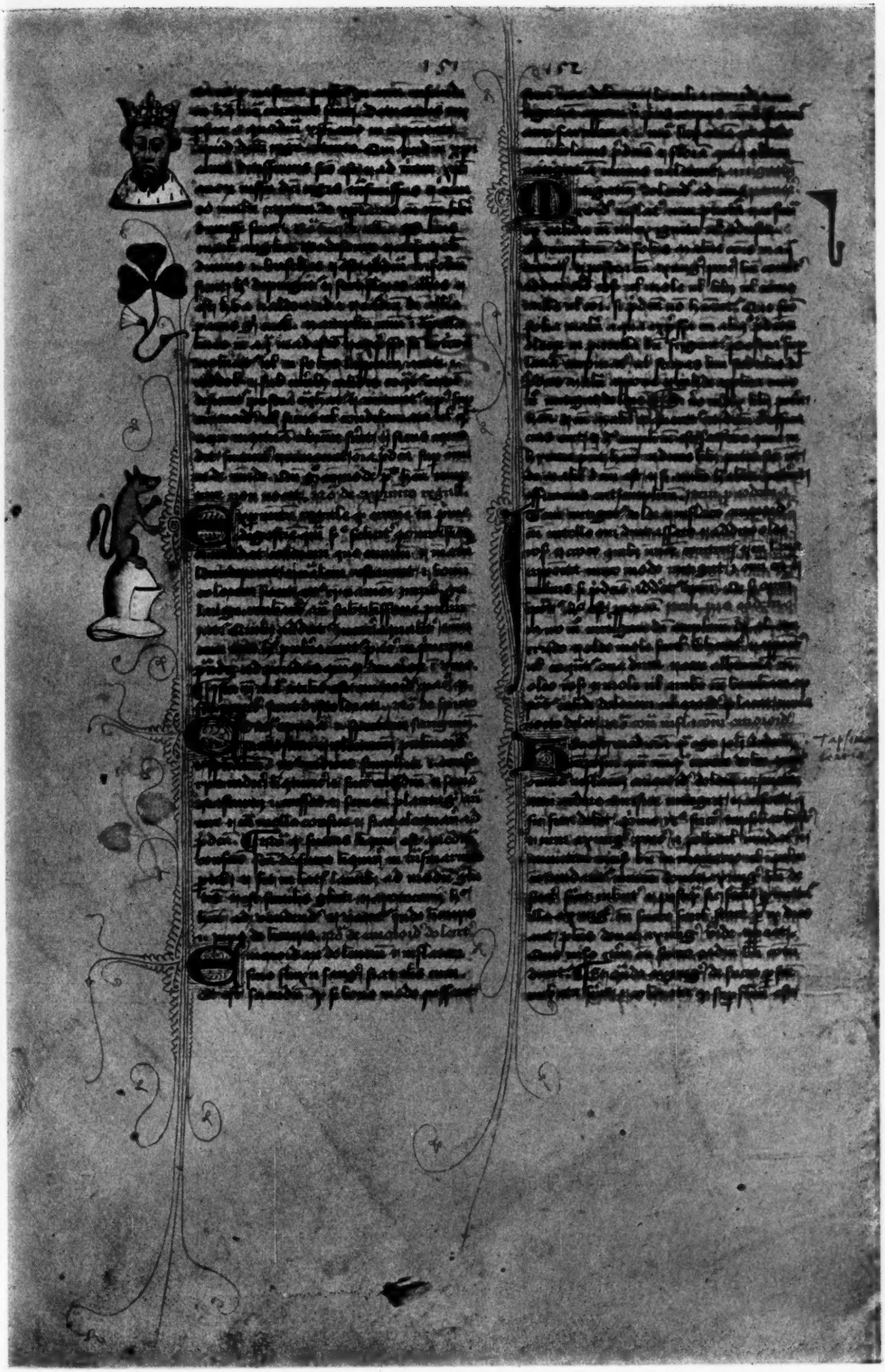
Item pro eodem.

Miro modo mitigare dicitur emplastrum de albumine oui crudo et oleo uiole simul uerberatis et appositis, uel coquuntur oua dura et eorum albuminia cum oleo rose et uiole, uel ambo, cum bombace apponatur calide dolorem emoroidarum placat et una nocte delet.

Nota contra inflacionem emoroidarum.

Hec est medicina quam ego Johannes Ardern composui quam nunquam carere volui quia omnem inflacionem emoroidarum dolorem ac puncturam cum ardore mirifice mitigat et aufert, que sic fieri debet. Primo, Recipe succum tapsibarbasti et cum axungia porci a pelliculis mundatum et minutim incisum bene in mortares, uel in pelui, contundantur inuicem donec axungia bene de succo fuerit imbuta et postquam sic feceris permittatur illa axungia cum succo simul stare per ix dies aut plus donec axungia viride appareat. Quo uiso iterum cum succo eadem bene contundatur. Et quando axungia de succo perfecte imbuta fuerit et colorata quod superfluum est.

Tapsiualem



*British Museum, Add. MS.* 29301, *f.* 40 *b*, *early XVth Century.*

*Plate II.*

42 expurgetur. Hoc est bis in hieme, et in vere quasi post quadragesimam semel, in estate semel vel pluries si necesse fuerit, nam eius beneficium nemo potest dinumerare. Quia sicut in necessitate pulcre adiuuat

de dolore lumborum — Ita eo vtentes a necessitate et dampno preseruat. Habeatur ergo in reuerencia.

*Dolor Renum* — Et quia dolor lumborum et renum habet vicinitatem cum colica et yliaca ideo hic aliqua experta *de doloribus renum* breuiter sunt notanda. Signa autem *doloris renum* sunt ha'c :—Dolor videlicet mansitat et in hoc differt a colica et yliaca quarum dolor est deambulatiuus per totum ventrem. Secundum signum est quod pes illius ex qua parte dolor oritur quandam patitur dormitacionem et vidi quendam qui non poterat mouere nec leuare a terra pedes nec tibias nec pedes ad se trahere quamuis contra ignem calidum positi concremarentur pro certo et tamen non paciebatur magnum dolorem et satis fuit sanus in reliquis partibus corporis et bene potuit commedere et bibere ac dormire, sed non ascellare pre ventris constipacione. Hec egritudo est valde difficilis curacionis si paciens transierit annos xl$^{a}$ secundum ypocratem Qui dicit Neferetici vltra xl$^{a}$ annos non curantur. Nichillominus tamen experiri possumus. Curacio ergo fit talis porro *si predictus* *sixtus* paciens *fortis fuerit* conuenientibus ceteris particularibus fiat flemina de sofena interiori eiusdem partis et si dolor fuerit ab ambabus partibus fiat de ambabus sofenis. Sofena vero non inuenta fiat de vena que dirigitur ad pollicem pedis. Deinde fiant clisteros vt in colica dictum est postea ponantur emplastra super renes. Et regula talis est quod medicus consideret qualitatem egritudinis vtrum sit ex causa calida vel frigida. Quod sic dinoscitur quod paciens sit colericus adustus cum calefaccione epatis in crocitate oculorum et quia in tempore ‡*proximi* totus comburitur et specialiter renes secundum Gordon tamen dolor renum frequenter accidit ex frigore. Dolor tamen interior prouenit ex calido. Quia frigiditas stupefacit et sensum membri remouet ex hoc quod spiritus ex renum opilatione non possunt ad membra inferiora penetrare quod autem sit in renibus opilatio patet in paroxismo est vrine retencio et cum curatur aduenit vrine multitudo et arena multa rubea insuper paroxismi et fine lunacionis.

1

2 nota

*fortis fuerit*

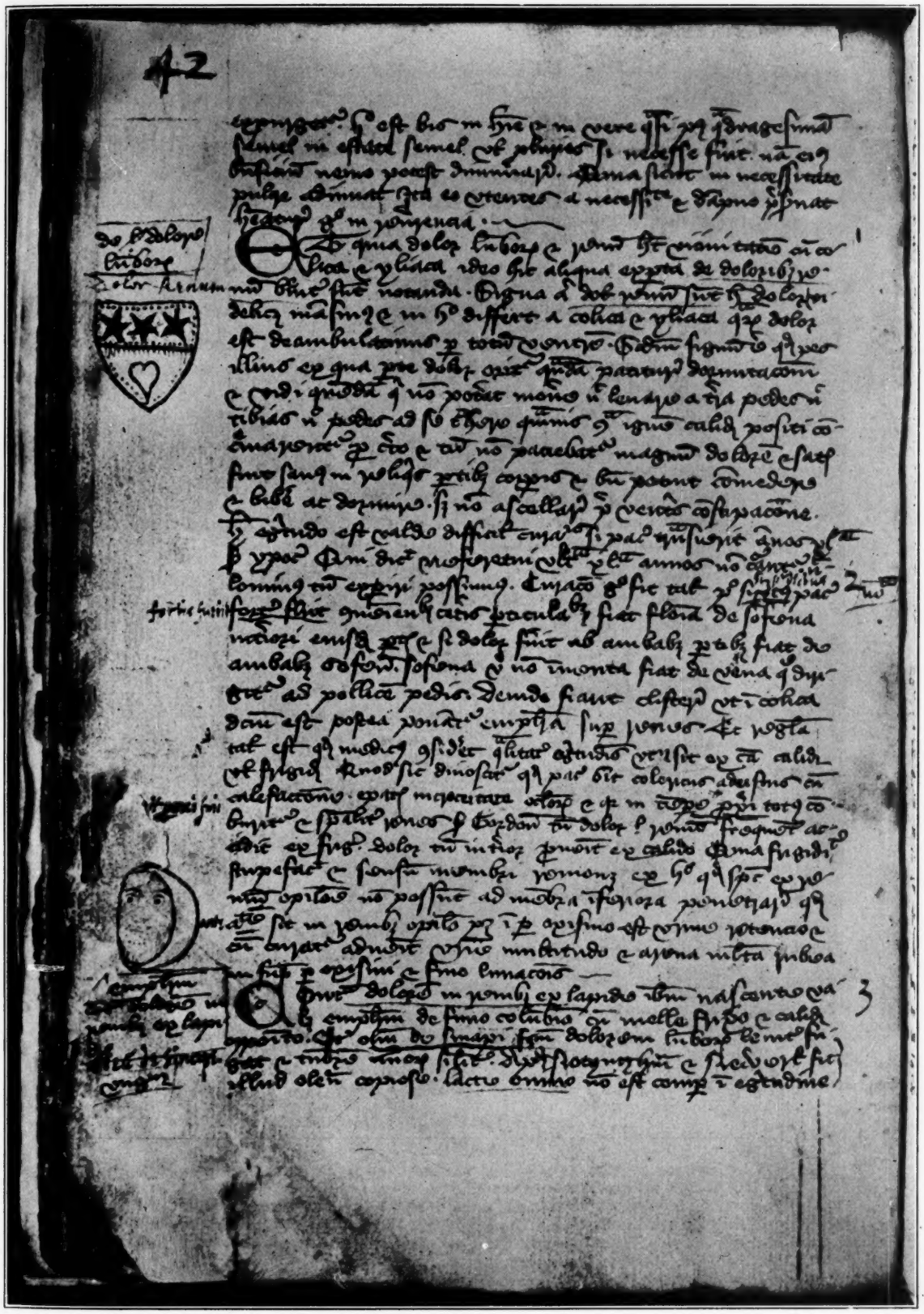
‡*paroxismi*

Emplastrum contra dolorem in renibus ex lapide

*oleum de sinape* vnguentum

Contra *dolorem in renibus ex lapide* ibidem nascente valet emplastrum de fimo columbino cum melle frixo et calido apposito. *Item oleum de sinapi* factum dolorem lumborum leuiter fugat et tumorem ani similiter. Apud Notyngham et Newerk fit illud oleum copiose. *Lacte ouino* non est compar in egritudine.

3



*British Museum, Harley MS. 5401, f. 21b, XVth Century.*

# ACHONDROPLASIA.

(*Chondrodystrophia Fœtalis.*)

By R. LAWFORD KNAGGS, London.

CONSULTING SURGEON TO THE LEEDS ROYAL INFIRMARY.

The condition known as achondroplasia is the consequence of *a defective evolution of the process of endochondral ossification, which is in evidence at a very early period of intra-uterine life.* The pre-ossification changes in the cartilage are probably influenced from the outset, and the chief stress of the disease falls upon the fœtus. This perversion of endochondral ossification is responsible for certain signs by which those in whom the affection is well marked can readily be recognized. These unfortunate individuals are: (1) Dwarfs; (2) Those with very short limbs, and a nearly normal-sized trunk; and (3) Those who have large heads and a peculiar facies.

At one time achondroplasia was confused with rickets, and the term '*fœtal rickets*' was applied to certain obscure affections of congenital origin which have since been differentiated into achondroplasia, osteogenesis imperfecta, and sporadic cretinism; but a certain haziness still attaches to it, for a doubt still lurks in many minds as to the existence of such a disease as intra-uterine rickets, though theoretically there is no reason why it should not occur, and certain interesting facts in favour of it are on record.

It was H. Muller, who, in 1860, recognized, as distinct from rickets, an affection which ran its whole course during intra-uterine life, and resulted in a characteristic appearance of the fœtus. Parrot, in 1878, gave to this intra-uterine affection the name 'achondroplasia', and in 1892 Kaufmann, who wrote more than one monograph on the subject, and studied its pathology minutely, called it 'chondro-dystrophia fœtalis'.* At first it was thought that the achondroplasic fœtus never survived, but was stillborn at the eighth month, but now it is well known that children and adults presenting unmistakable signs of the disease are to be seen occasionally going about their ordinary avocations. Moreover, we know from archæological records that the achondroplasic individual has been recognized from the earliest times.†

There seems to be no reason why those cases that are born alive and survive the first year should not live for the ordinary term of human life, and some are mentioned by Emerson as having attained to the ages of 84, 66, 48, and 41 years respectively.

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* For further information on the history, *see* Marie, *Presse méd.*, 1900, July 14, 17.

† An interesting note by George Pernet (*Brit. Jour. Child. Dis.*, i, 17) refers to a " number of glazed earthenware figures (statuettes) most of them of some shade of green " in the British Museum (Third Egyptian Room). These portray the deformity of achondroplasia, and are representations of the Egyptian god Ptah-Seker-Asar, the triune God of the Resurrection (Dr. E. A. Budge). Jansen, in his monograph," Achondroplasia; its Nature and Cause ", gives an illustration of the god Bes—the Egyptian god of sexual intercourse (Elliot-Smith).

It is quite clear that there are different degrees of the disorder. In many cases it is so pronounced that the fœtus dies in utero and is aborted; whilst those that survive may present various grades of the characteristic deformity. Since a very large proportion of achondroplasiacs are stillborn it happens that the pathology of the disease has been chiefly studied in the fœtus, but for clinical purposes there has been no lack of material both in children and adults. In this article, therefore, unless it is otherwise stated, the clinical side of the disease is drawn from living cases, and the description of the skeletal changes and the histology are taken from the fœtus.

## THE CLINICAL FEATURES OF ACHONDROPLASIA.

The following section has been largely culled from the excellent accounts given by Pierre Marie,[1] Souques,[2] and others. The typical achondroplasiac at birth has a normal-sized body, exceedingly short limbs, and a large head with a very characteristic depression at the root of the nose. His stunted stature becomes increasingly evident during childhood and adolescence, and is largely due to the shortness, and the feeble growth in length, of the lower extremities (*Fig.* 1). The child walks at the proper time, teeth appear at the usual age, the rolls of fat which disfigure the limbs slowly disappear, but growth always remains retarded, and when the child reaches adult age it has for a long time been an unmistakable dwarf.

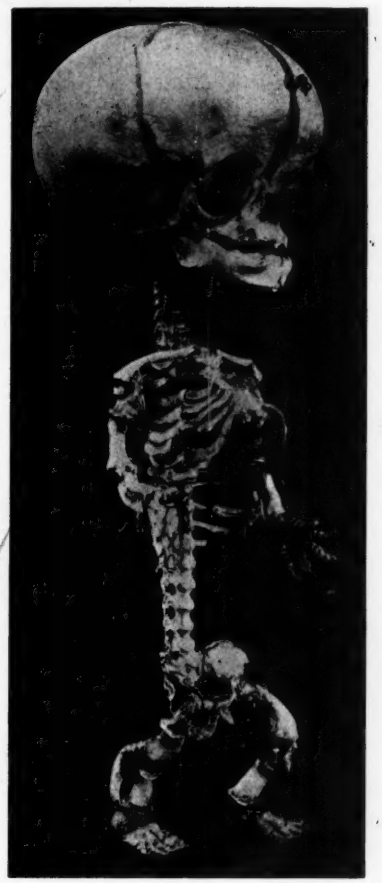

Fig. 1.—Photograph of the skeleton of an achondroplasic fœtus. Liston's case. The frontispiece in Liston's *Elements of Surgery* (2nd edit., 1840) is clearly a drawing from the specimen. (*R.C.S. Museum*, No. 4157·1.)

The height of the male achondroplasic dwarf varies between 115 and 125 cm. Of P. Marie's two cases, Anatole, age 40, was 122 cm., and Claudius, age 18, was 107·5 cm. The shortest recorded are Broeckh's case, a woman, age 23, who measured 97 cm., and Bailly's, age 27, 104 cm.

**The Shortness of the Limbs (Micromelia).**—This gives rise to some striking peculiarities. When the arms are hanging down, the tips of the fingers reach only to the great trochanters or the iliac crests, whereas in the normal person they extend to the lower third of the thigh. The unusual brevity of the lower limbs trenches on the ridiculous, and, owing to this obtrusive deformity, the central point of the whole body, which should be at the umbilicus, is placed much higher—as high sometimes as the ensiform cartilage. The proximal segment in all the limbs

is most affected (rhizomelia), the arm often being shorter than the forearm, and the thigh than the leg, a ratio which is the opposite of the normal. The hands and feet are also shortened; but less, relatively, than the longer bones (*Fig.* 2).

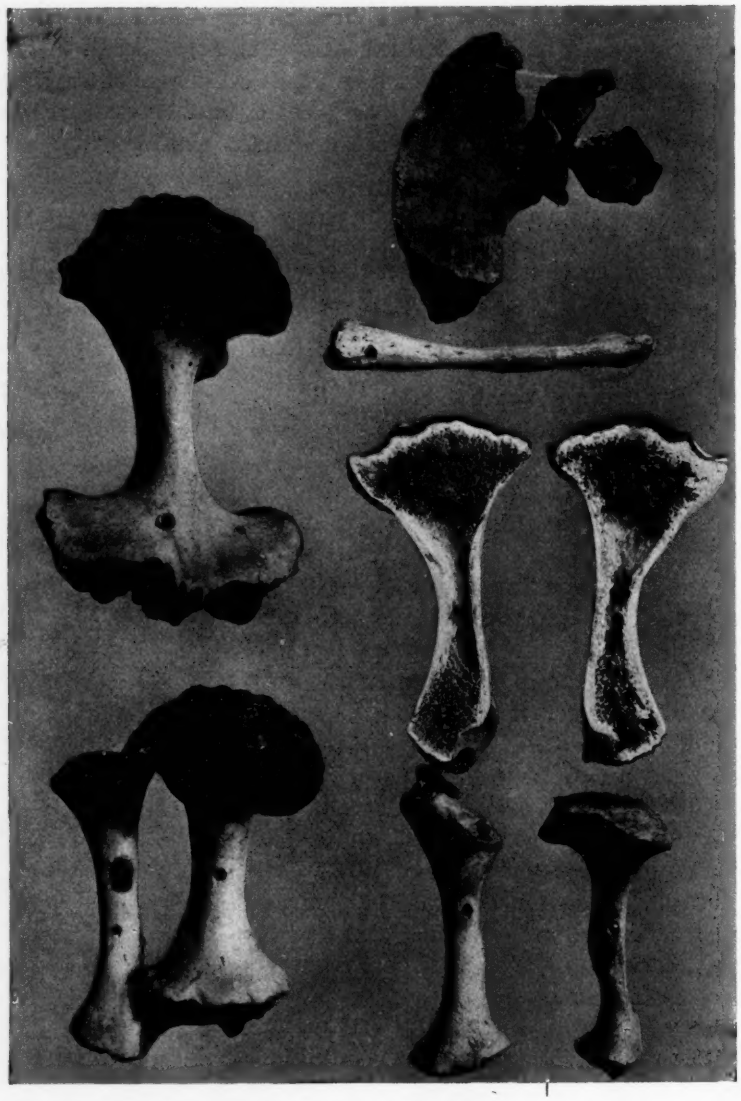

FIG. 2.—The illustration shows the short shafts with abruptly expanded ends characteristic of achondroplasia. The bones of the upper extremity are on the right, and those of the lower on the left. Note the relative length of the clavicle, which, being largely a membrane bone, shows no shortening. (*R.C.S. Museum*, No. 4163·1.)

These macerated bones are those of a full-time fœtus, born dead, and were taken from a very unusual case described by Shattock (*Pathol. Soc. Trans.*, 1880, xxxii, 369). The specimen is entitled in the Catalogue, "Achondroplasia and Rickets"; and the justification for the latter part of the title appears to lie in the porous thickened condition of the pelvis and cranial bones (No. 4163.3), and of portions of the surface of some of the long bones.

The effect of the shortening may be judged from Turner's[3] account of a 'very intelligent' girl, age 10, who was unable to wash herself thoroughly

or to attend to herself in the ordinary calls of nature, took three hours to dress, was entirely debarred from active games, and could only write with great difficulty owing to the characteristic deformity of the fingers and the hand.

**The Achondroplasic Hand.**—This is short and broad, and the fingers are all of equal length. When the hand is stretched out with the palm flat the points of the fingers diverge from one another, the divergence being most marked between the ring and middle fingers. Marie called it the 'main en trident', and various explanations of the way in which the divergence is produced have been suggested.*

**Curvature of the Limbs.**—The short limbs are often curved, especially the lower ones; as a rule, however, this is not caused by bending of the bones, which in adults are usually straight, but by an angular displacement of the two segments at the knee, the articular surface of the tibia looking slightly outwards.

**Abnormal Position of the Head of the Fibula.**—In this interesting peculiarity the head of the fibula lies on the same level as the head of the tibia and appears to form part of the knee-joint, but as it does not present an abnormal facet it probably does not lie within the joint cavity. Souques points out that this position of the head of the fibula is normal in the eighth week of fœtal life, but in health the tibia develops more rapidly than the fibula, whose upper end eventually comes to lie at some distance from the joint.

**The Nearly Normal Trunk.**—The short limbs contrast strangely with the large and apparently normal trunk to which they belong. The latter is said to have, as a whole, a more or less normal growth, and looks like the body of an adult to which are fitted four limbs suitable in point of length to a young child. In Marie's two adult cases the bodies were much longer than that of a boy of 8 who was of intermediate height and used for comparison. But though the limbs are very disproportionate to the trunk, yet the growth of the latter is also retarded, though to a much smaller degree than that of the limbs. Turner's case, a girl of 10 years, was shorter than her sister of $3\frac{1}{2}$, but when sitting side by side their heads were on the same level. There can be no doubt that when the younger girl reached the age of 10, her body would considerably exceed in length that of her achondroplasic sister at the same age.

Balme and Reid[4] found the distance from the occipital protuberance to the tip of the coccyx in their case of a boy, age 12, to be $20\frac{3}{4}$in., whilst in a normal boy of similar age it was 24 in. This impaired growth of the spine, which occasionally is very striking, is in harmony with the retarded development of the ossific centres of the bodies in the stillborn fœtus. These are always much smaller than those of a full-time healthy infant, or even of a fœtus some weeks or even months before birth (*see Figs.* 13 and 14).

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* Ettore Levi attributed the divergence of the fingers to articular hypotonus, common in achondroplasiacs, which leads to imperfect adaptation of articular surfaces; Bertolotti, to the condyloid surface of the first phalanx not being on a horizontal plane, and causing a deviation on the depressed side; and Souques, possibly to bony changes at the distal extremity of the first phalanx.

**The Head** (*Figs.* 3 and 4).—The head is absolutely and relatively large—a fronto-occipital circumference of 60 cm. ($21\frac{3}{8}$ in.) is not rare, and is occasionally exceeded. In shape it is rounded and markedly brachycephalic, a condition which is the outcome of the peculiar development of the basis cranii and the supra-occipital bone. Its transverse diameter is greatly increased and the frontal and parietal bosses are prominent (*Fig.* 4). The face is broadened, but less than the cranium, and the short nose presents a characteristic depression or indentation at its root. In some cases the whole nasal region may be flattened (*Fig.* 3). These latter signs are related to the abnormal development of the cartilaginous part of the basis cranii in front of the foramen

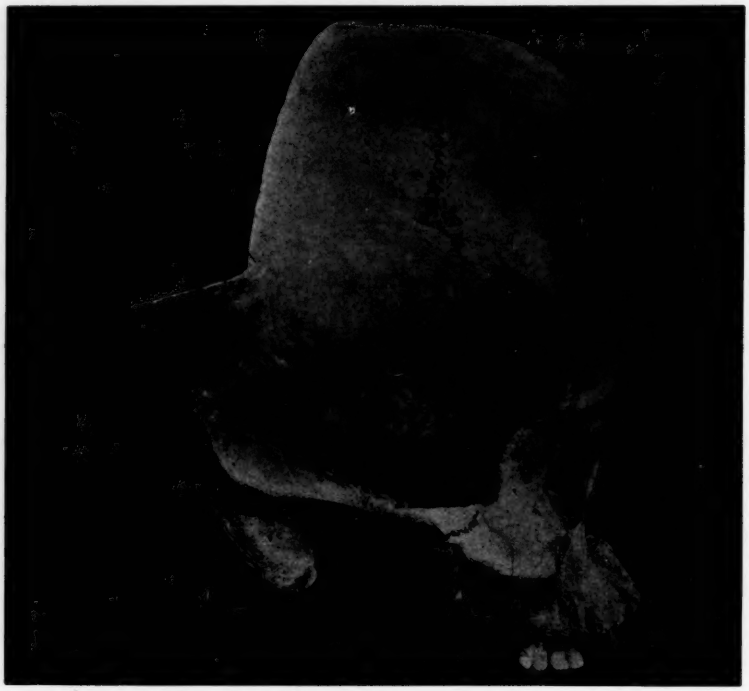

FIG. 3.—An adult ancient Egyptian (XVIIIth Dynasty) achondroplasic skull. (Professor Flinders Petrie. *R.C.S. Museum, Gen. Path. Sect.*, No. 96·2.)

" The face is flat ; the nasal bones are small pyramidal nodules, the spine of the frontal appearing at their lower ends ; the ascending maxillary processes project very slightly in front of the lower border of the orbits. The premaxillary region of the upper jaw is drawn upwards ". " There is a profound aplasia of all those bones laid down over the anterior part of the ethmoid or trabecular cartilages ".

The condition present is exactly like that seen in the bull-dog. The nasal bones and ascending parts of the maxillary of the bull-dog are arrested and thickened ; the premaxillary part of the palate is tilted upward owing to the circumnasal atrophy. (Keith, *Jour. of Anat.*, xlvii, 198.)

magnum. When the flattening of the face is marked the alveolar process of the upper jaw is pushed forward, owing to the alveolar growth which takes place to accommodate the teeth. The alveolar border has gone on growing, whilst the face has not. The protrusion of the upper alveolar processes and the prominence of the lower jaw result in prognathism ; a condition which is well shown in the skull of Graham's case preserved in the Anatomical

Museum at Birmingham, and in the ancient Egyptian skull shown in *Fig.* 3.

**The Spine.**—*A kyphotic curve* in the dorsal or dorsolumbar region is often present in the fœtus as well as in adults [*see* J. Thomson's cases in Bowlby and Andrewes' *Surgical Pathology,* 7th ed., *Fig.* 84; Symington and Alexis Thomson's case (fœtus), and No. 95·1, R.C.S. Museum, Gen. Pathol. Section (fœtus)]; but it is not the rule in the latter, the back being more commonly described as flat.

On the other hand, *lordosis* is a characteristic feature, even when kyphosis is present higher up. It was very pronounced in Dr. J. Thomson's cases, in

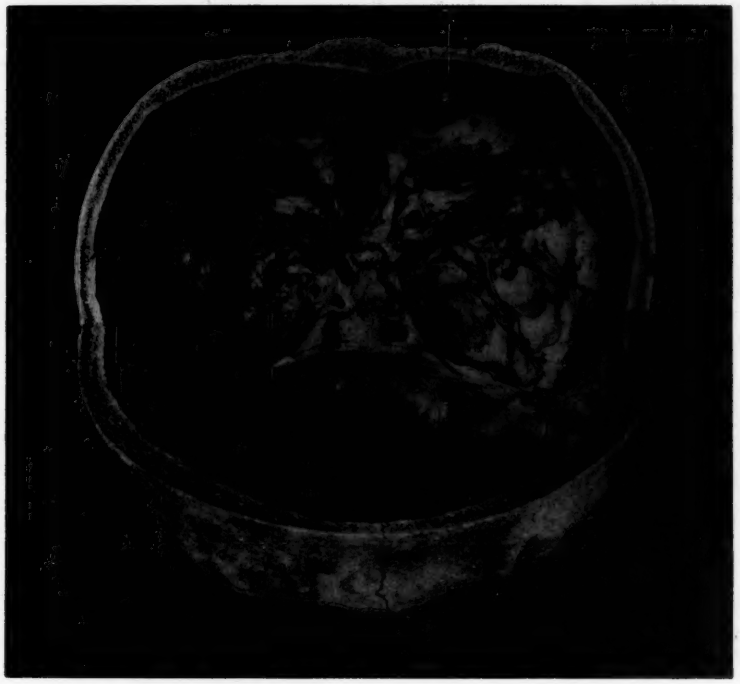

FIG. 4.—The interior of the ancient adult achondroplasic skull shown in *Fig.* 3. The internal auditory meatuses are the same distance apart as at birth (24 mm. or $\frac{15}{16}$ in.) The posterior fossa retains infantile dimensions. The occipital bone has the same linear dimensions as the occipital of the normal child at birth (superimposed). The pituitary fossa is distinctly small (12 mm. a.-p. by 10 mm. trans.). The sagittal and lambdoid sutures are obliterated; the coronal simple in formation and open. Owing to non-expansion of the posterior fossa the cerebellum has lifted the lateral sinuses and tentorium up to or above the lambdoid suture (Keith, *Jour. Anat.*). Note the small petrous bones and the unusual shortness and the increased width of the skull, giving it a globular shape. (*R.C.S. Museum, Gen. Path. Sect.,* No. 96·2.)

which the forward movements of the spine (in picking up) were very limited. There is some uncertainty as to the true explanation of this lordosis or lumbosacral curve. It is attributed by many to the tilting of the sacrum, the hollow of which is carried backwards and upwards. Keyser[5] made the important observation that it did not disappear on placing the patient on his back and elevating the legs, as it does when the hip-joints are rigidly fixed in hip-joint disease. But if an adult achondroplasic innominate bone is compared with

a normal one, the ilium, and especially its upper expanded portion, is seen to be not only much smaller than the corresponding part in the healthy bone, but to be definitely more poorly developed than its own ischial and pubic portions. The acetabulum is normally placed in relation to the ischium and pubes, but, owing to this poor development of the ilium, its posterior border impinges on the great sacrosciatic notch. The femoral heads consequently are placed further back than usual. This supports Poynton's[6] view, which attributes the curve to the attachment of the femora to the skeleton in a plane posterior to that of the normal individual: "the nates accordingly are unduly prominent, the sacrum is less vertical, and this, coupled with the prominence of the buttocks, gives the superficial impression that there is lordosis of the spine. This is not, however, the case, the vertebral column being if anything less curved than normal."

**The Pelvis.**—The tilting of the sacrum, whatever its cause, is a considerable factor in the contraction of the pelvic inlet, whilst the outlet is increased by it. Also, as might be expected, the achondroplasic pelvis is diminished in all its diameters. Consequently, the disease is of very obvious moment in women when they become pregnant. Not only are abnormal presentations often observed, and prolapse of the cord frequently seen, but normal labour is nearly always impossible. Many recorded cases illustrate this. Porak[7] removed a fœtus at full term from a woman, age 27, by cephalotripsy, and at a subsequent pregnancy an infant showing signs of the same disease was removed by Cæsarean section. Charpentier was able to deliver by forceps a child which lived only a few hours. Bailly's case was delivered by craniotomy, and the symphysis ruptured during traction. Again, in Curvail's case Cæsarean section was performed.

**Intellect.**—The achrondroplasiac approaches the normal intellectually. Not infrequently they are lively and amusing, but in a considerable number intelligence is reduced, or they are backward for their age. One of Marie's cases at 18 was like a quiet moderately reserved boy, and had not learned to read because his memory was so defective. Some of this backwardness may be due to their difficulty in associating with other children in consequence of their deformity. But their power of control over themselves is deficient, and they are reputed to be vain, boastful, excitable, fond of drink, and sometimes lascivious (*see* note on the god Bes, p. 10).

**Sexual Development.**—This is usually normal, though sometimes behindhand, as in Marie's case just mentioned, which showed only doubtful evidence of puberty.

**Muscular Development, etc.**—The achondroplasic dwarf is very muscular and excels in feats of strength, a feature which, coupled with an eccentric appearance and oftentimes amusing behaviour, leads to their frequent employment in theatres and circuses, for which cretins would be unsuitable owing to their mental defects. He is also a favourite character in fiction in the capacity of a court fool or a nobleman's minion.

**Union of the Epiphyses.**—Radiography has shown that there is great variation in the time at which the epiphyses may be completely ossified. Though bony union may occur at the normal time, and sometimes takes place prematurely, as in Mery and Labbé's[8] case of a boy of 12, in whom ossification

was abnormally advanced and the epiphyseal lines early obliterated, yet it is apt to be delayed considerably, and in some cases it is never completed. Thus, union was not perfect in one of Marie's cases at the age of 40, and in some of the bones of Graham's[9] case "the epiphyseal lines which should be last obliterated are still distinctly visible, though this subject was 46 years of age".

**Post-natal Onset.**—Emerson[10] refers to various cases in which children apparently normal at birth developed achondroplasia later. In one which he had excellent opportunities to study the child was born normal, but had an acute disease in infancy resembling rickets. Signs of the deformity began about the age of 4. He sums up the position with regard to such cases in this way: "We may therefore believe that achondroplasia is a disease that may develop after birth. Its symptoms are those of an acute illness which considerably retards the child's normal development. Flabbiness, tenderness of the flesh, and sweating of the head are prominent symptoms. The teething, talking, sitting up, and walking are all retarded, and the relative micromelia is first noticed after this illness. But some of these are the classic symptoms of rickets."

A post-natal onset must be very doubtful. It would be difficult to eliminate a slight grade of the disorder, and, on the other hand, in very badly nourished children who are the subjects of rickets endochondral proliferation is practically at a standstill, and explains the very stunted growth sometimes seen.[11]

**Heredity.**—The majority of achondroplasiacs are sporadic, but the affection shows an unmistakable hereditary tendency. The evidence is, of course, limited, for the achondroplasic fœtus is in most cases stillborn, and only in slight degrees of the disorder can the female produce a living child without suitable operative procedures. Some interesting examples have been recorded. Porak's case, already mentioned, was delivered of an achondroplasic fœtus by Cæsarean section. In like manner Baldwin[12] saved an affected child which lived nine and a half months. In Boekh's case the great-great-grandfather (*trisaieul*) and the sister of the patient were achondroplasiacs; and J. H. Porter[13] records 3 cases—a father and two sons—with a remarkable family history. The disease occurred in six members of a family of three generations; all were males, and not a single female was produced in those three generations.

## THE MACROSCOPIC CHANGES IN THE BONES.

These are very conspicuous in:—

1. *The long bones of the extremities*, and
2. *The base of the skull*; and less noticeable in
3. *The vertebral column*; and
4. *The thorax.*

### 1. THE LONG BONES OF THE EXTREMITIES.

In the stillborn fœtus in which the disease is pronounced, these bones present a fantastic appearance. Large, somewhat rounded, and, in comparison

with normal epiphyses, more or less misshapen masses of cartilage are united by a very short diaphysis. A great part of the length of a bone, often more than half, is formed by its two epiphyses, which, in consequence of their relative bulk, make the connecting shaft appear very insignificant (*Figs.* 9, 10). The several bones are much shorter than in corresponding ones in the normal fœtus, and a particular bone may be more readily identified by its position than by its shape. The proximal bones are more affected than the distal ones. In the humerus and femur the shortening amounts to more than a

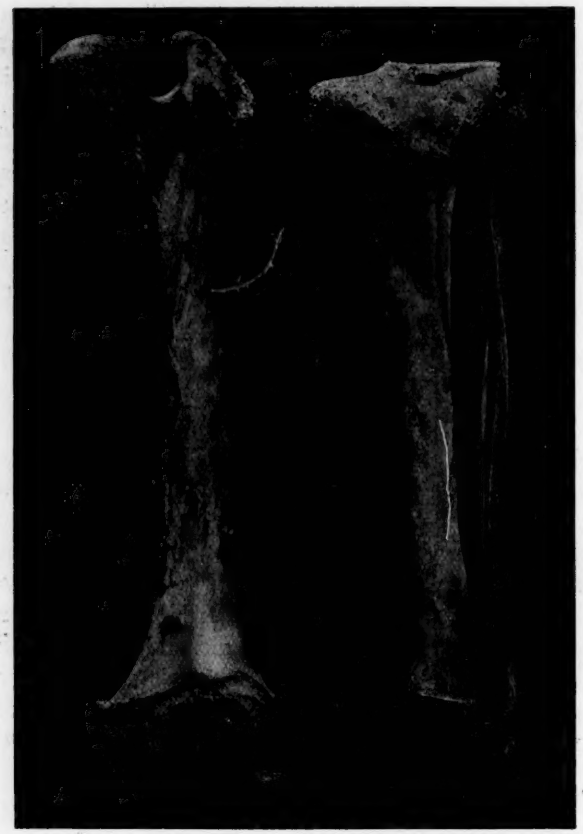

FIG. 5.—The bones of the lower extremity of an achondroplasic adult negro. Length of femur 10⅜ in., tibia 9¼ in., fibula 9⅜ in. The fibula has been adjusted to the tibia lower down than it should have been; its head should be on a level with the head of the tibia. Note the grooves marking the seats of junction of the lower epiphyses to the shafts. (Dr. Christopherson's specimen.) (*R.C.S. Museum Stores.*)]

third of their length, and in the forearm and leg to a quarter at least. The deficient length-growth of the shafts is responsible for this stunting. The ends of the diaphysis spread out abruptly to adapt themselves to the much wider epiphyses, and so the bones in many cases have a dumb-bell shape (*see Fig.* 2). The shafts are thick for their short length, and more clumsily modelled than those of a healthy fœtus, and the bones may show bends or angles situated at the juxta-epiphysial regions. Such angles are attributed

to an absence of growth at one end of the epiphysial junction line, the cause of which will be explained later.

In adults (*Figs.* 5–8) the normal curves may be exaggerated, but the ends of the bones are large and broad, and terminate abruptly at their junction with the shafts, whilst the muscular insertions are strongly marked. In a vertical section the cartilage of the epiphysis is firm, and in some bones a thin white fibrous layer can be seen in it, bordering its line of junction with the diaphysis. The cortex of the shaft is thick and hard. It may even invade the central area to a greater or less

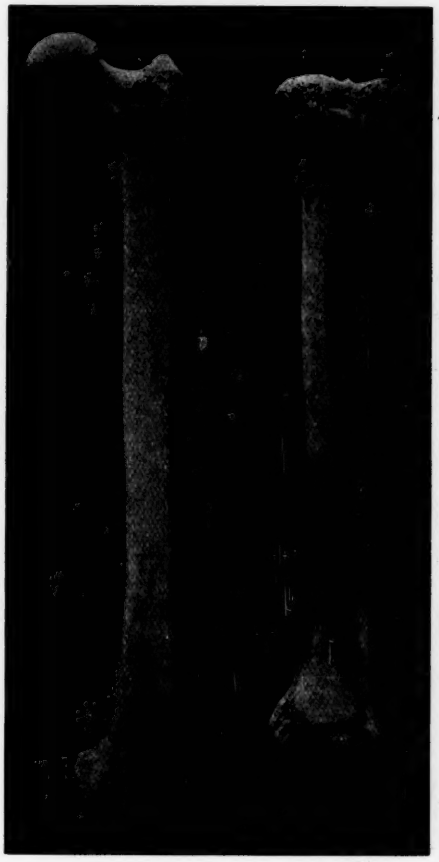

FIG. 6.—Normal adult bones. Length of femur 17⅞ in., tibia 15 in., fibula 14¼ in. (*R.C.S. Museum, Anatomical Section.*)

FIG. 7.—The bones of the upper extremity from the same skeleton as *Fig.* 5. Length of humerus 7 in., ulna 6⅝ in., radius 6 in. All the epiphyses are united.

FIG. 8.—Normal adult bones. Length of humerus 13⅛ in., ulna 10¼ in., radius 9½ in. (*R.C.S. Museum, Anatomical Section.*)

extent, so that occasionally the shaft is formed almost entirely of compact bone. But in some instances a wedge-shaped area of endochondral bone may be recognized extending for a short distance into the diaphysis from the epiphysial junction (*Fig.* 9). At the ends of the bone the compact tissue tends to overlap the adjacent portion of the epiphysis, which then appears slightly morticed into the diaphysis.

FIG. 9.—Section of an achondroplasic femur—taken from the same case as *Fig.* 12. Note the thick compact bone of the diaphysis and its tendency to overlap the cartilaginous epiphyses, which are relatively very large. The marrow is apparently normal throughout, and shows no fat cells. (Dr. Greenfield's section.) (×2¼.) (*All the photographs are by Dr. G. H. Rodman.*)

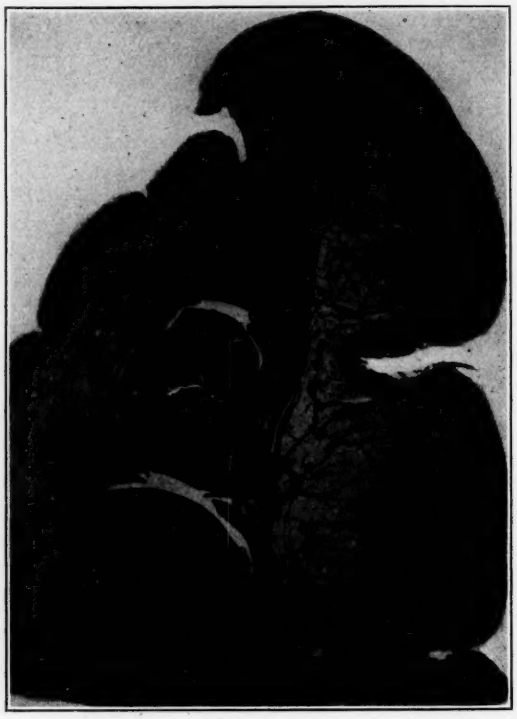

FIG. 10.—Section of an achondroplasic great toe—from the same case as *Fig.* 12. (Dr. Greenfield's section.) (×3½.)

"*The scapula* is smaller than normal, especially in its vertical diameter, and its glenoid cavity is apparently too small for the head of the humerus. In hyperplastic cases the cartilaginous part may be bulky and massive, especially the marginal epiphysis, and the ventral surface deeply concave." (Kaufmann.)

### 2. THE BASE OF THE SKULL.

The important changes in the skull occur in that part of it which is developed in cartilage, especially the axial part of the base formed by the body of the sphenoid, the basi-occipital, the ex-occipitals, and the supra-occipital. The petrous bones, also of cartilaginous origin, are often unusually small, a condition which is marked in Graham's case

(Birmingham Anatomical Museum), and in the Egyptian skull (*see Fig.* 4).

In the *normal* fœtus the bar of cartilage from which the body of the sphenoid and the basi-occipital develop presents three centres of ossification at birth—the pre-sphenoid separated by a synchondrosis from the post-sphenoid, which again is separated by another synchondrosis from the basi-occipital (*Fig.* 11). The pre-sphenoid and post-sphenoid centres fuse by the end of the first year, but the post-sphenoid and basi-occipital centres do not blend till the age of 25, when bony ankylosis of the basi-cranial axis is complete (Cunningham).

The occipital bone at birth consists of four parts, the interparietal and supra-occipital combined, the basi-occipital, and the two ex-occipitals, one on either side of the foramen magnum. The interparietal portion forms in membrane, but the rest of the bone grows in cartilage. The two ex-occipitals are separated from the basi-occipital by synchondroses in front, and in like manner from the supra-occipital behind. Fusion of the two ex-occipitals with the basi-occipital takes place about the fourth or fifth year, and with the supra-occipital by the third year or earlier. Thus, in the normal child at birth all these different ossific centres are distinct, and are separated by intervening cartilage.

In the *achondroplasic fœtus*, whether stillborn at full time, or even earlier, quite a different condition is present. Instead of three separate centres, the pre- and post-sphenoid and basi-occipital nuclei are represented by a single mass of bone which, without doubt, is produced by the fusion or synostosis of the three nuclei. Moreover, the ex-occipitals are frequently fused with the supra-occipital, and often with the basi-occipital, or, if this has not occurred, there is no intervening cartilage, which in a normal case would be several millimetres thick.

As a consequence of the interruption of growth caused by this early fusion of the nuclei (*see* p. 37), the 'os tribasilare'—the name given by Virchow to the part of the basi-cranial axis represented by the three ossific centres in front of the foramen magnum—is much shorter than the normal (*Fig.* 12), whilst premature union of the ex-occipitals results in a small and often cordate foramen magnum, which cannot grow larger, but whose sides grow steeper owing to the continued periosteal ossification.*

This remarkable departure from the normal in the development of the base of the skull accounts for the following peculiarities which distinguish the achondroplasic skull and its contents :—

*a.* The typical *achondroplasic cranium* is round and globular, being markedly broader and shorter than normal. Its peculiar brachycephalic

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* In Symington and Thomson's carefully recorded case of a full-time fœtus, the distance from the foramen magnum to the anterior edge of the pre-sphenoid was 2·4 cm., whilst in a normal fœtus used for comparison it was 3·6 cm. The anteroposterior diameter of the foramen magnum was 0·65 cm., and in the normal 2·2 cm.

The interparietal part of the occipital bone was well formed, but the parts developed from cartilage were much smaller than normal. The supra-occipital was ossified to the ex-occipitals, but osseous union was not present between the ex-occipitals and the basi-occipital, though there was no intervening cartilage. Synostosis was complete in the case from which *Fig.* 12 was taken, and also in one of Sir Thomas Barlow's cases (*Univ. Coll. Hosp. Museum*, No. 633), but in another (No. 636) it was absent.

shape is due to the way in which the membranous bones of the vault accommodate themselves to the growth of the brain. This organ, which is cramped by the want of development of the base of the skull, obtains the room it needs by causing expansion of the vault.

So, in Symington and Thomson's case all four fontanelles were enlarged, and the vertical plates of the frontal bulged forwards, whilst the two halves

FIG. 11.—Normal skull at birth. Sagittal section. The pre- and post-sphenoid and the basi-occipital nuclei have been outlined in black. The dura mater at the base is indicated by a white line. The loose coronal and lambdoid sutures can be made out. A, Atlas; L, Lambdoid suture. ($\times \frac{2}{3}$.) (*R.C.S. Museum Stores.*)

of that bone and the two parietals were further apart than usual. In rare instances early union of some sutures may lead to considerable peculiarity of shape owing to the brain's expanding in the only direction that may be possible. But sometimes the sutures of the vault are not so loose as in the normal infant. Thus, in the fœtus from which *Fig.* 12 was taken they were quite firm, and by transmitted light it could be seen that this was due to developing synostosis.*

*b.* The basal changes in front of the foramen magnum are responsible for the deep depression of the root of the nose, or the flattening of the face, which are characteristic marks of the achondroplasic facies. The effect of

* Some interesting points are found in the skull of Graham's case in the Birmingham Anatomical Museum; it is that of an adult, age 46.

1. The maximum length is 192 mm.; the maximum breadth 163 mm. The cranial index equals $\frac{163 \times 100}{192} = 84{\cdot}8$. An index over 80 indicates a brachycephalic skull.

2. The orbital plates of the frontal bone are raised up and bulge into the anterior fossa,

the synostosis of the os tribasilare is increased by another alteration which has not yet been mentioned. The clivus—that portion of the base which intervenes between the foramen magnum and the dorsum sellæ and represents the intracranial surface of the posterior portion of the tribasilar bone—has a much more vertical position and is also narrower than in the normal individual.*

FIG. 12.—Skull of an achondroplasic fœtus—stillborn. Sagittal section. The three nuclei in front of the occipital foramen are fused into a single piece of bone (black outline). Note the almost vertical direction of the clivus, the less obtuse sphenoidal angle, and the displaced tentorium. The coronal and lambdoid sutures can be seen to be rigid and synostosed. The small size of the foramen magnum is very noticeable when compared with that in *Fig.* 11—anteroposteriorly it measures 6·5 mm.; in the normal skull (*Fig.* 11) 11 mm. The measurement from the ethmoid spine to the anterior edge of the foramen magnum is 42·5 mm.; in the normal skull (*Fig.* 11) it is 58 mm. A, Atlas; L, Lambdoid suture. ($\times\frac{2}{3}$.)

leaving a deep gutter corresponding to the cribriform plate of the ethmoid. This condition was probably caused by the growth of the eyeballs, which could not enlarge in a downward direction as the face had stopped growing.

3. The clivus is almost vertical. The petrous bones are very small and the distance between the two internal auditory meati is $1\frac{3}{4}$ in., which may be compared with $2\frac{1}{4}$ in. in a normal skull, and points to the retardation of lateral growth in this axial portion of the basis cranii.

4. The zygomatic fossa is remarkably short anteroposteriorly (*see also Fig.* 3). This is evidently connected with the want of growth of the face and its failure to be normally projected.

5. The jugular foramina are unusually small (defective growth of the bones) and the mastoid foramina are very large (to provide a collateral channel to carry off the blood from the lateral sinuses).

* The sphenoidal angle is the angle which the basi-occipital part of the basi-cranial axis makes with the pre- and post-sphenoid portion, i.e., which the ascending portion of the axis makes with the horizontal portion in front of it. This angle in the normal fœtus, in the

Partly owing to the arrested growth and partly to the altered position of this portion of the basi-cranial axis, the cartilaginous parts in front of it, which are concerned in the development of the nasomaxillary region, are not sufficiently pushed forward. Hence arises the depressed nasal root, or, if the nose as a whole fails to be projected, the flattened face.*

*c.* The changes in the occipital bone account for: (i) *The synostosis, and small size of the foramen magnum*; and (ii) *The practical elimination of the cerebellar fossa* owing to reduced size of the supra-occipital bone.

The internal occipital protuberance is only a very short distance from the posterior margin of the foramen magnum and—owing to displacement and occasional alteration in the shape of the cerebellum—the tentorium, and with it the lateral sinuses, have a much higher attachment to the interior of the skull. Thus the torcular Herophili was 6·3 cm. from the foramen magnum in Symington and Thomson's case instead of 2·5 cm. which is the normal, and the lateral sinuses and the tentorial attachment corresponded to the lambdoid suture (*see Figs.* 4 and 12).

*d.* The *brain and medulla* also show some changes :—

i. The lower part of the medulla is notably diminished in size when the foramen magnum is very small, and this fact points to the *smallness and rigidity of that aperture being responsible for the premature death of the achondroplasic fœtus.*

ii. The long axis of the medulla and pons, owing to the steep clivus, instead of being directed upwards and forwards, is more nearly vertical, or even inclined upwards and backwards, and the upper border of the pons comes to lie considerably above the dorsum sellæ to which it should normally correspond.

iii. The smallness of the occiput below the internal occipital protuberance, by practically abolishing the cerebellar fossa, leads to striking changes in the cerebellum. In Symington and Thomson's case it was flattened, and its long axis directed from the foramen magnum upwards and backwards. In one of Barlow's[15] cases the cerebellum was pushed forwards and had grown upwards so that it was 'nested' in the cerebrum, which covered it more than in a healthy fœtus. The adaptive alterations of the tentorial attachment just described are associated with these changes.

### 3. THE VERTEBRAL COLUMN.

This was the normal length in the case so often quoted (Symington and Thomson's), and each body was practically of normal dimensions, but the ossifying nuclei in the centres were only half the normal size. The small

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period from three months to birth, attains to 155°, and from then to puberty gradually increases (flattens out). In achondroplasia, owing to the more vertical condition of the clivus, it is much less obtuse than this, and the increased curve which its diminution indicates has been described by Virchow as a kyphosis of the base of the skull. (Kaufmann, " Untersuchungen ueber die sogennante fœtale Rachitis (Chondrodystrophia fœtalis).")

* Kaufmann[14] points out that the marked retraction of the nose root, or the flattening of the face, may be present in rare cases in which the base is quite soft and cartilaginous, or in which there may be no shortening of the tribasilar. He ascribes this to defective growth of the cartilaginous parts in front of the tribasilar.

size of the nuclei is a constant feature in the spine of the achondroplasic fœtus and is very striking when compared with those of the healthy stillborn infant (*Figs.* 13, 14; *see also Figs.* 11, 12). Sometimes there is a premature synostosis of the body and arch of the upper cervical vertebræ similar to that of the modified vertebra which surrounds the foramen magnum (Bruns and Kolisko).

*The pelvis* has already been described (p. 16).

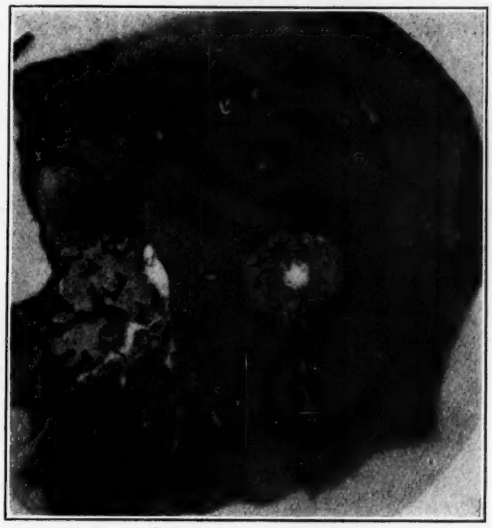

FIG. 13.—Section through a lumbar vertebra of a stillborn achondroplasic fœtus—same case as *Fig.* 12. (×7.)

#### 4. THE THORAX.

The ribs show evidence of arrested development, and may be less than half the normal length at birth, whilst the costal cartilages are of average size. In Symington and Thomson's case they joined the cartilages at an angle and so produced a furrow or depression at the costochondral junctions. The beading often present at these junctions is due to the end of the rib expanding to receive the termination of the cartilage.

FIG. 14.—To compare with *Fig.* 13. Section through a lumbar vertebra of a normal full-time fœtus. (× 7.)

* * * *

Kaufmann[16] has distinguished three forms of achondroplasia—the hypoplastic, the hyperplastic, and the malacic—according as the cartilage destined to form rows shows no recognizable change, or with normal consistence shows an exaggerated undisciplined growth, or is softened. In the hypoplastic form the epiphyses are rather smaller than normal and the length of the diaphysis is less than half the normal. In the hyperplastic variety the epiphyses are thick, broad, and mushroom-shaped on short diaphyses, but not so short as in the hypoplastic form. In the former (hypoplastic form) the bones are frequently bent, and thick for their short length; in the latter the diaphyses are straight. It is in the hypoplastic type that there is such vigorous periosteal activity, i.e., where the bones are very short, and the fibrous layer grows into the epiphysis from the periosteum. In the malacic form the epiphysial cartilage is abnormally soft, and unsuitable for physiological growth and development.

## HISTOLOGY.

The most striking fact in the histology of achondroplasia is a greatly diminished proliferation of cartilage cells, which is sometimes so extreme that the process may be practically non-existent. This process, when it occurs at the epiphysial ends of the long bones, is responsible for their growth in length, and it is in consequence of its failure to evolve, or of its interruption if it has once been established, that the dwarfing which characterizes the disease results. If the long bones of an adult achondroplasiac are compared with those of a fœtus afflicted with the same disease, it is evident that a considerable growth in length has taken place since birth, presumably upon more or less normal lines, but not as much as would have occurred in an ordinary individual. Two possible explanations of this post-natal growth suggest themselves: (1) The disease may be active only during intra-uterine life, and as its activity diminishes normal ossification may assert itself; or (2) The disease may be of a mild character from the start, and persist so long as growth continues.

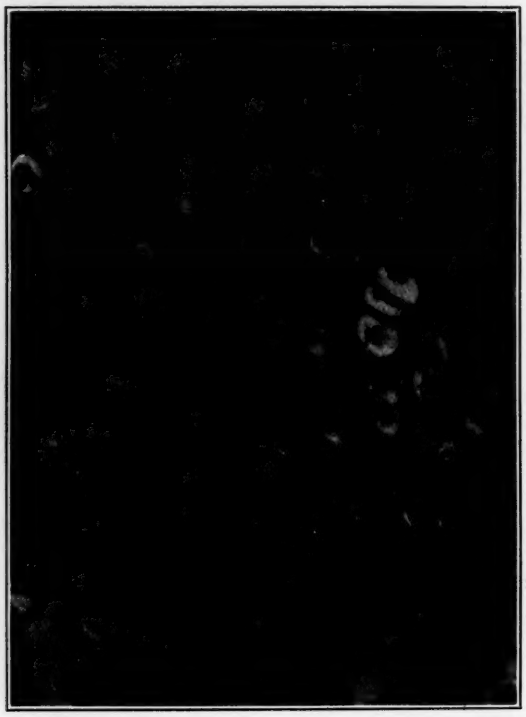

FIG. 15.—High-power view from the same femur as *Fig.* 9, showing bone formation preceded by calcification of cartilage; also the shallow zone of cartilage proliferation. (×270.)

The second explanation is probably the correct one. Severe degrees of the disorder result in the death of the fœtus, and the presumption is that those cases which survive and reach adolescence represent a less pronounced type of the disease. Even these show different grades of the affection, and in some it may be so little marked as to escape recognition except after careful investigation.

*The microscopical appearances seen at the junction of an epiphysis with the diaphysis* in a fœtal achondroplasic long bone are of two distinct types, neither of which is normal in that situation. The first exemplifies ossification through an intermediate stage of calcification of cartilage; the second, ossification by metaplasia of a fibrous tissue that has either developed by metaplasia from the cartilage or arisen by an extension into the cartilage from the periosteum.

If the junction line be traced from side to side under a low power, there will be seen at one end, or sometimes at both ends, a narrow layer of *compact bone* reaching inwards for some distance and blending externally with the

compact tissue of the diaphysis. The rest of the boundary area is occupied by cartilage showing a narrow zone of cell proliferation which is undergoing calcification preliminary to the formation of *cancellous tissue* (*Figs.* 15, 16).

Ossification through calcification of cartilage is the chief process; the formation of the compact bony layer is a secondary one; it is not always present; it is partial in most instances (*Fig.* 17); but it sometimes seems to involve the whole length of the line, especially in the small bones such as the phalanges, and then cartilage cell proliferation and swelling of the cells is seen to have come to a complete standstill (*Fig.* 23).

The appearances seen in the area where cartilage undergoes calcification are the following:—

1. There is a complete absence of the palisade-like line of provisional calcification which is such a marked feature of normal ossification at the epiphysial lines.

2. The zone of proliferating cartilage cells is very narrow. *There is no arrangement of the cartilage cells in columns.* The absence of this 'row formation' has been emphasized by all writers on the subject as the histological feature of greatest importance (*Figs.* 15, 16, 17). In the shallow zone of proliferating cartilage the cells are numerous. They are also large in contradistinction to those in the quiescent cartilage close to, and may be crowded here and there into little groups; or they may be scattered freely, irregularly, and somewhat closely, but without any attempt at arrangement; though in some sections two or three cells may be in contact in such a way as to suggest an incipient row.

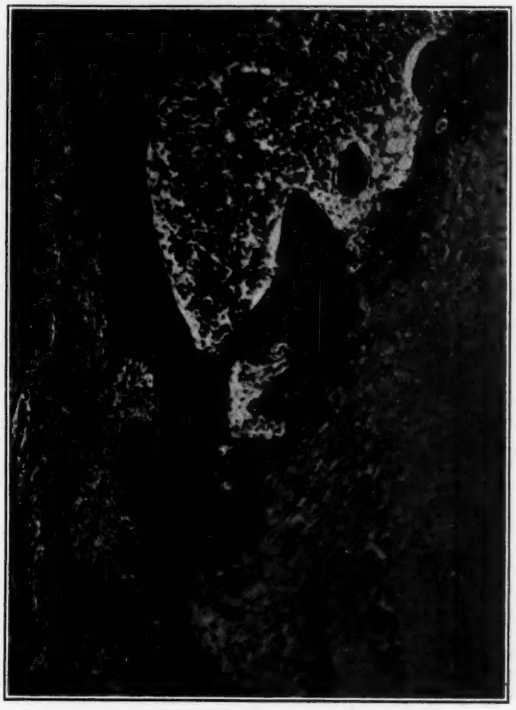

FIG. 16.—One corner of the epiphysial border of the terminal phalanx shown in *Fig.* 10, showing the very slight degree of cartilage proliferation, the swelling of the cells, and the calcification of the cartilage. The faint tendency of the cartilage to assume a fibrous form close to the periosteum can be recognized. The calcified cartilage is stained purple and the true bone added in places on its medullary surface yellowish. The difference can be detected in the photograph by the deeper shading of the calcified cartilage. (×85.)

3. The ground substance of the cartilage in which the enlarged cells lie is seen to be undergoing calcification on the side adjacent to the diaphysis, and trabeculæ containing cartilage cells become outlined by the breaking up of the calcified zone by medullary spaces (*Fig.* 15). Osteoblasts, next, border the notched margins of these trabeculæ, and laminated bone is laid upon the calcified cartilaginous foundation. A little deeper in the medullary tissue the

trabeculæ are composed entirely of laminated bone, and their origin in calcified cartilage cannot be made out, or only in some instances.*

A very different process is seen to lead to the production of the *compact layer of bone* which advances inwards from the periphery of the line of junction. As the boundary region is approached the cells in the cartilage become more numerous and crowded, but not so large and definite as those in the

FIG. 17.—Lower epiphysial junction—not far from the part shown in *Fig.* 20. Ossification by metaplasia of fibrous tissue on the right, and by calcification of cartilage on the left. (×85.)

* The relation that *normal ossification* at an epiphysial line bears to endochondral ossification in achondroplasia calls for some explanation. The significance of the 'row formation' is that it points to rapid proliferation of cartilage cells. Nature has evolved this careful packing of the superabundant cells at the epiphysial ends of the long bones, and the orderly method of their delivery to the bone-forming department, to enable the limited space at her disposal to be utilized to the best economical advantage. In the small cartilage bones, and in the basis cranii in the healthy fœtus, the area of cartilage proliferation and 'row formation' around the ossifying nuclei is neither so deep nor so crowded with cells as at the epiphyso-diaphysial junctions. Provision for length growth is not called for to the same extent. This no doubt explains why the region of provisional calcification in these bones takes the form of a zone of calcified cartilage instead of the palisade-like arrangement which marks the epiphysial lines in the long bones. As this calcified zone is broken up by the advancing medullary tissue, irregular plates are formed and upon them laminated bone is laid down by osteoblasts. The cartilaginous foundation of many laminated trabeculæ is evident at a considerable distance from the zone of calcified cartilage, and sometimes all over the section of a complete nucleus.

The appearances seen in the nucleus of an *achondroplasic vertebra* are like those in normal small cartilage bones except that there is a slighter degree of proliferation of the cartilage cells, and an absence of the 'row formation'. And they are similar in achondroplasic long bones at those parts of the epiphysial junctions where ossification is preceded by calcification of cartilage.

Evidently histology does not point to a fundamental difference between chondral ossification in achondroplasia and in the normal fœtus. The various appearances may be explained by the relative activity or inactivity of cartilage proliferation, and the deciding factor between the 'palisade' formation and the zone of calcified cartilage attributed to the amount of matrix persisting between the cells in which lime salts can be deposited. Obviously this must be much less where the cells are crowded and closely packed together as at the epiphysial lines.

area where the cartilage is undergoing calcification. They elongate and can in many sections be traced gradually into fibrous tissue; and on the diaphysial side of this fibrous layer compact tissue is formed by metaplasia from it (*Figs.* 18, 19, 21). The layer of compact bone is thin and of variable length, and on its medullary side it is fringed with osteoblasts and joined, more particularly near its periosteal end, by trabeculæ of periosteal formation (*Fig.* 23). Where it occurs it walls off the medullary spaces from the cartilage.

The fibrous tissue takes the form of a strand or layer which in many bones can be recognized by the naked eye on the epiphysial side of the line of junction. A great deal has been written about it, and its origin. It is believed to be derived from the periosteum, and in proof of this the presence in its meshes of "islands of cartilage and fat cells" has been cited (Emerson). It has been attributed by one authority to an ingrowth from the periosteum, and by another to an infolding of that membrane. Undoubtedly the tract blends with the periosteum at its outer end, and has the appearance of an offshoot from it sweeping round the corner of the diaphysis to lie *in the cartilage*. But the equally evident transition of the cartilage cells from the quiescent area till they merge into the fibrous layer would suggest that the cartilage is undergoing a retrogressive change (metaplasia to fibrous tissue), which is most pronounced at the marginal part of the boundary area, possibly owing to the influence exerted by the propinquity of the periosteum. Whatever the true explanation of this fibrous change in the cartilage may be, there is no doubt about its importance. It is the preliminary stage in the formation of a compact bony lid to the end of the diaphysis, which must effectually prevent any further growth in length. Usually it is only seen partially developed, and if it is present on one side only, growth ceases on that side, and a bend or angle forms at the juxta-epiphysial region in consequence of the continuance of a certain amount of growth on the other. If it occurs on the inner side at the upper epiphysis of the femur, the outer side of the diaphysis growing upwards may tilt over the head and neck and produce coxa vara.

FIG. 18.—High-power view of a portion of a section shown in *Fig.* 17, showing ossification by metaplasia of fibrous tissue almost certainly developing from the cartilage. (×270.)

*The formation of bone by the periosteum* takes place in the normal way,

but the process is unusually active, no doubt, because it has to make up, as far as it can, for the failure of the cartilaginous ossification. This activity is a noticeable feature of the hypoplastic variety described by Kaufmann.[14] The cortex of the diaphysis consequently is abnormally thick, and the compact bone may even invade and obliterate the central cavity. At the ends it tends to grow over the line of junction and to embrace the adjoining part of the epiphysis. In some cases, on the other hand, rarefaction may occur in the central part of the diaphysis to such an extent (osteoporosis interna) that it may predispose to fracture. In Kaufmann's case of chondrodystrophia hyperplastica there were fractures of several of the long bones which it is possible occurred during delivery by turning. The central cavities were large and the periosteal bone formation was much less than in hypoplastic cases.

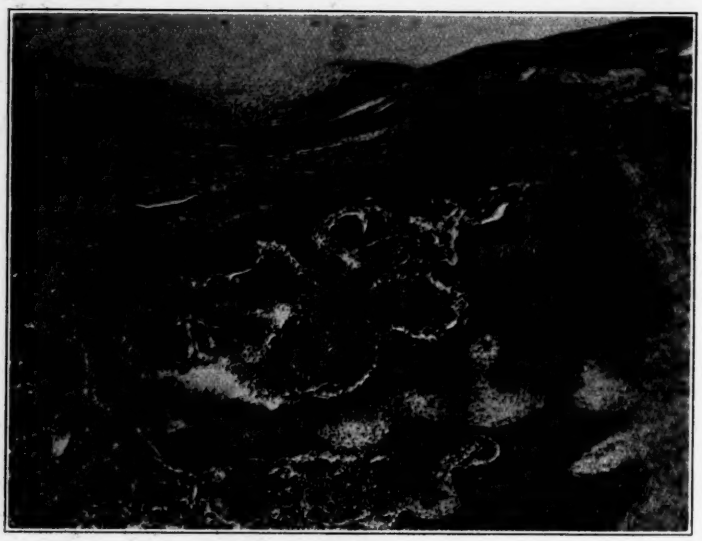

FIG. 19.—The right-hand corner of the junction line of the upper epiphysis of the femur shown in *Fig.* 9. The fibrous tissue is seen sweeping round the bone from the periosteum. The transition from quiescent cartilage to fibrous tissue is noticeable on the epiphysial side of the layer of compact bone, which borders the fibrous tissue on its diaphysial side. (×50.)

**The Marrow.**—Those who have described the marrow speak of it as very cellular and not lacking in blood-vessels. Giant cells and evidence of resorption may or may not be seen. The illustrations show the usual cellular character of a medulla, and the presence of very few fat cells. Evidently the marrow in achondroplasia is not wanting in vitality, and its behaviour in connection with the periosteal ossification and even the limited endochondral ossification shows that its functional capacity, so far as bone building is concerned, is good. There is consequently no reason to think that the absence of cartilage cell proliferation is a secondary manifestation dependent upon an unhealthy condition of the marrow.

## PATHOGENESIS.

**The Time of Onset and the Duration of the Disease.**—To follow the various points that arise in the pathogenesis of achondroplasia it is important

to determine the period of intra-uterine life at which the disease begins. Normally centres of ossification appear in the cartilaginous shafts of the bones of the limbs in the second month; first in the upper extremity and later in the lower. It is clear, therefore, that the affection cannot begin before that date. But even if the conditions responsible for initiating the disorder were present then, there would be a further delay before endochondral ossification advanced to the extent of defining the epiphysial ends, and until that took place the normal equilibrium between growth of cartilage and bone formation would not be reached, and the commencement of rapid length-growth, the absence of which is the first clear evidence of the disease, would not occur.

The earlier writers believed that achondroplasia ran its whole course in utero. Parrot regarded it "as a dystrophy of the cartilage concerned in the first skeleton formation during the period from the third to the sixth month of fœtal life" (Emerson); and Symington and Thomson thought that "those bones escaped which, though formed in cartilage, remain entirely or mainly cartilaginous till an advanced period of fœtal life so that their general growth is quite independent of endochondral ossification"; but even such bones will show microscopic evidence of the disease when ossific nuclei are present. It may be assumed, therefore, that "the pathological process is active, and the conditions that induce it present, from between the second and third month of intra-uterine life, and that on account of the early onset" only certain bones appear to the naked eye to be affected.*

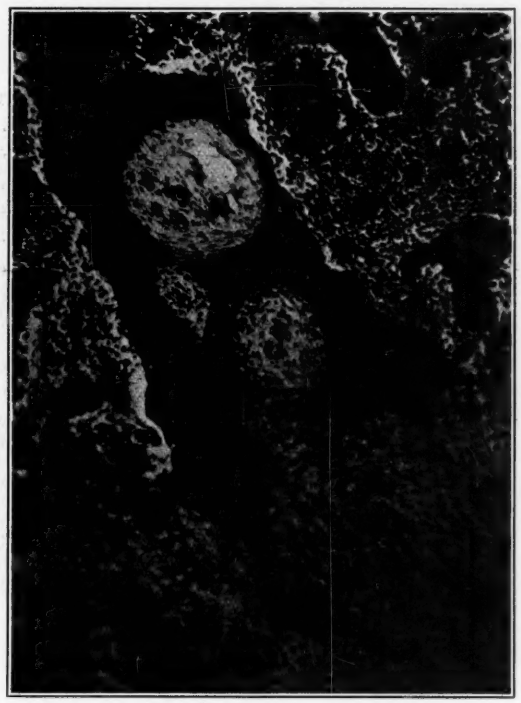

FIG. 20.—From the lower epiphysial junction of the same femur as *Fig.* 9 —about the centre—showing a trabecula forming from fibrous tissue interposed in the line of calcifying cartilage. The large circular medullary space shows an osteoid bordering. (×85.)

But if the onset of the affection can be traced to the latter part of the second or the beginning of the third month, its duration is not so easy to determine. In the previous section it has been presumed that the disorder probably persists as long as growth goes on, but it does not follow that it is of the same intensity all the time. The possibility of recovery, connected no doubt with the disappearance of the causal condition, must be recognized, but there is nothing

* The presence of the notochord or its relics in one or two of Kaufmann's cases was thought to trace the onset of the disturbance of growth to about the second month. But this is not of much value, for traces of the notochord are not infrequently found long after they should have disappeared.

to show that it occurs. On the other hand, an occasional clinical fact may point to a relapse or an aggravation, or even suggest a late onset. Thus Turner's case (*see* p. 12)—a girl of 10, in whom the deformities were not noticed at birth—did not grow during the five years before the case was recorded. She wore the same frocks as she did when between 4 and 5 years old.

### THE CORRELATION OF ACHONDROPLASIA AND OSTEOGENESIS IMPERFECTA.

There are some suggestive points of resemblance between these affections both in their intra-uterine origin and their post-natal course. In achondroplasia the proliferation of the cells at the growing epiphysial region of the cartilage is seriously at fault, and the length-growth of the bone almost

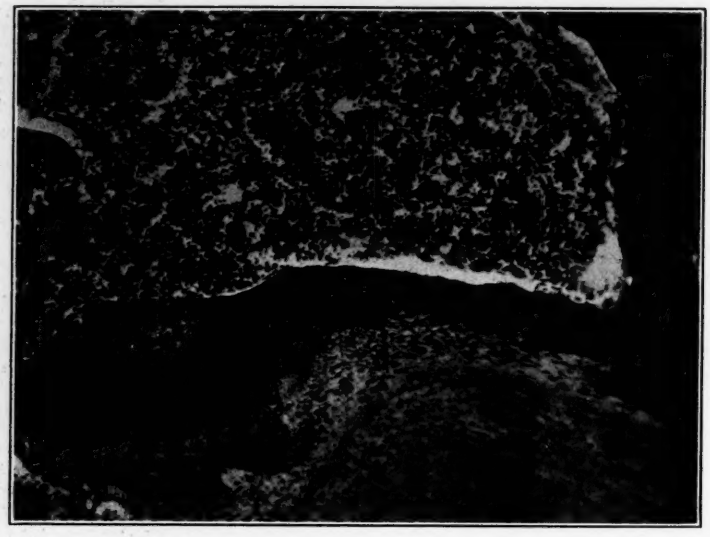

FIG. 21.—The other corner of the epiphysial border shown in *Fig.* 16. Showing a layer of compact bone forming from a thin strand of fibrous tissue which is continuous to the right of the picture with the periosteum. (×85.)

abolished. In osteogenesis imperfecta the stress falls on the other great bone-forming tissue. The cells formed from the periosteum are not of the normal type, being more akin to cartilage than osteoblasts; and they function badly, producing a delicate and crumbling kind of bone of inferior vitality and strength.

A critical inquiry into the correlation of the two disorders would be out of place here, but it is important to note that in both the changes begin in the embryo and are probably present from the very commencement of the pre-ossification changes. The thought that the phenomena of both diseases may prove to depend upon somewhat similar or allied causes is very natural, and it would not seem impossible for some rare case to present conditions suggestive of both. Such a case is the following.

**Hektoen's Case.**[17]—This was "a short-limbed dwarf supposed to be 45 years of age, who supported himself by selling lead pencils and chewing-gum from a roller. He was presumably of fair intelligence; with a fibroid thyroid without any demonstrable thyro-iodine; with a relatively large cranial vault, made up of 172 Wormian bones, but without any evidence of premature ossification of the synchondroses at the base of the skull; with marked curvatures of the spinal column; with old fracture of many of the ribs and of the left humerus; with great deformity of the pelvis; with curvatures of most of the long bones which are relatively short, the articular ends being swollen into irregular globular masses; with a general and pronounced osteoporosis; and with the absence of trident fingers" (*Fig.* 22).

FIG. 22.—Hektoen's case. The skeleton shows some of the features of osteogenesis imperfecta and some of those of achondroplasia—*see* text, (From *Amer. Jour. Med. Sci.*, 1903, cxxv, 756, *Fig.* 5.)

Discussing the explanation of this peculiar case Hektoen writes: "Cretinism or rickets or osteomalacia in all probability is not wholly responsible for the changes present in the skeleton. Taking all the facts and conditions into consideration it must be acknowledged that the case presents some features best explainable on the score of osteogenesis imperfecta and others best explainable on the score of chondrodystrophy fœtalis, but as for reaching definite conclusions it is unfortunate that the picture of either of these two diseases, as now understood, is incomplete in some essential particular; and furthermore nothing is known of this man's earlier history."

### ACHONDROPLASIA AND THE INTERNAL GLANDULAR SECRETIONS.

Certain pathological facts have done much to stimulate interest in the physiology of bone growth. The increased size of the bones and the delayed obliteration of the epiphysial lines in eunuchs (*see R.C.S. Museum, Pathol. Sect., Disorders of Growth, Specimen* No. 4846·1,), the dependence of cretinism upon diseased and atrophic thyroids, and the association of acromegaly and gigantism with pituitary conditions, caused attention to be focused more particularly upon the testis, the thyroid body, and the pituitary gland, but other glands believed to provide internal secretions—such as the parathyroids and the pineal—may be said to be 'under observation'.

The following sketch of some recent researches suggests that the pathogenesis of achondroplasia may prove to be intimately concerned with the abnormal functioning of one or more of them.

**The So-called Cretinous Calf.**—A good deal of literature has been devoted to the 'cretinous calf' and its connection with achondroplasia. Crew[18] tells us that the Irish Dexter is a breed of the smallest cattle in the British Isles, with a short broad head and short legs, two points (brachycephaly and micromelia) which have an important relation to the subject. A Dexter mated with a Dexter bull invariably results in a proportion of dead, misshapen calves. The period of gestation is approximately 284 days, and it can be foretold when a pregnancy will terminate in a 'bull-dog' fœtus. About the third or fourth month great abdominal distention sets in, and this is followed by a flow of amniotic fluid. Then the flow ceases and the cycle is repeated. Following one of these discharges the fœtus is aborted.

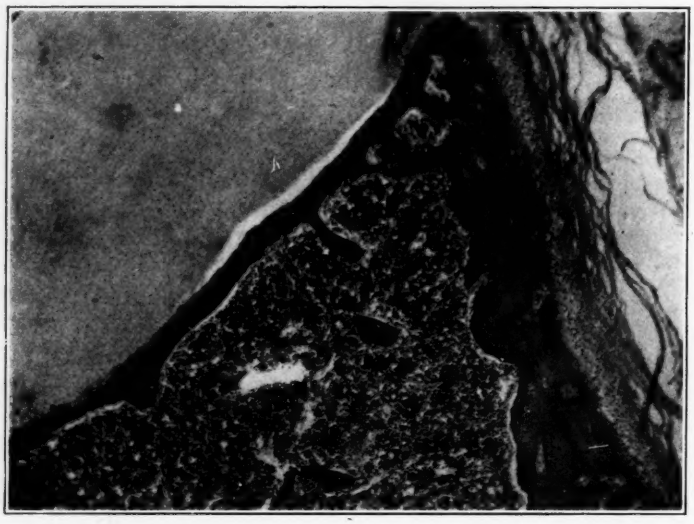

FIG. 23.—One half of the distal epiphysial border of the metatarsal bone shown in *Fig.* 10). The compact border occupies nearly the whole width in this section, but not in another one. (This section was taken near the periphery of the bone.) There is a complete absence of proliferation of the cartilage on the epiphysial side of the compact layer except in the vicinity of a short interruption to the left of and outside the picture. At each corner the first sign of an ingrowth (?) from the periosteum is in evidence. There is very little bone formation on the medullary side of the compact layer. The medulla shows a number of scattered fat-cells; and the cross-section of a considerable vessel lies outside the picture. (×50.)

The above cycle is not invariable. The first sign may be the abortion of a dead monstrous calf, or a premature labour necessitating operative measures.*

The Dexter 'bull-dog' fœtus is very much smaller than the normal fœtus of the same age. Its death is associated with severe fœtal anasarca (fœtal dropsy) in the case of the earlier abortions, and in the majority of the later ones; and with very difficult labour owing to the shape of the fœtal head. When there is hydramnios there is fœtal anasarca, the fœtus being a water-logged and shapeless mass, and the abdominal wall devoid of skin over a

* The puerperium differs from that following a normal calf: (1) The placenta comes away in small fragments instead of being thrown off complete in three or four hours; and (2) The abortion of the monstrous calf does not interrupt the production of milk, whereas the abortion of a fœtus other than a 'bull-dog' does immediately.

circular area based upon the umbilical cord. The anasarca present in the majority of cases is common in cattle, and invariably associated with death and abortion in the sixth or seventh month. Its cause is not known, but in the case of the 'bull-dog' fœtus, it is always associated with hydramnios—both probably being due to the same cause.

Crew came to the conclusion "that the conditions found in the 'bull-dog' calf are such as are found in the clinical and pathological entity known as achondroplasia. It would seem that the exhibition type Dexter is a low-grade achondroplast, and that the 'bull-dog' calf produced by the mating of two such individuals is a high-grade achondroplast exhibiting the classical features of the condition in a most pronounced form".

C. G. Seligmann's[19] investigations led him to the conclusion that the changes in the 'bull-dog' calf were due to a deficiency in the thyroid secretion, and that the monsters were, in truth, cretinous calves. He demonstrated the ineffectiveness of their thyroid secretion by intravenous injections of fresh extracts of the gland (50 per cent glycerin) into animals. No fall of blood-pressure, or a very slight one, occurred, and this contrasted with the effect produced when a similar extract, made from a normal newly-born Dexter-Kerry, was employed.

Hogben and Crew,[20] turning to account an interesting biological fact, came to quite an opposite opinion. They employed the axolotl test. This larva of the Mexican salamander, when kept in tanks, preserves its larval form indefinitely, but feeding it with potent thyroid tissue causes it to undergo rapid metamorphosis into a fully developed terrestrial amblystoma. By this test it was impossible to obtain evidence of active secretion in the normal fœtal thyroid earlier than three months in the sheep and four months in the ox, and practically similar results were obtained in the case of the 'bull-dog' fœtus.

The result of an inquiry into the functioning of the *posterior lobe of the pituitary* of the monstrous calf is described by Crew in his communication to the Royal Society. An injection of a minute quantity of posterior-lobe extract into a frog "previously kept under those conditions which conduce to skin pallor" is followed by a very characteristic and marked darkening due to a conspicuous expansion of the melanophores (Hogben and Winton). This result is produced by the posterior-lobe extract of normal cattle at the beginning of the third month, but that of the 'bull-dog' fœtus of six months gave a very doubtful reaction, and from one of four months a still more doubtful one. A year or two ago the anterior and posterior lobes were regarded as entirely unconnected glands with different functions, but there is now a tendency to look upon them as one gland, although the distribution of the colloid is unequal in the various parts (Rendle Short[21]). There is reason, therefore, to believe that the melanophore test indicates the functioning of the pituitary as a whole and not of the posterior lobe only. Crew's researches show, therefore :—

1. That the thyroid becomes active in the 'bull-dog' fœtus about the same time as in the normal cattle fœtus, and some time after ossification changes have begun.

2. That the pituitary becomes functionally active in the normal fœtus at the beginning of the third month, i.e., some time before the thyroid does,

and about the time when ossification changes are beginning at the ends of the long bones.

3. That in the 'bull-dog' fœtus the pituitary is late in beginning to function, and is largely inactive even at the sixth month.

From these facts it follows that the monstrous calf is not associated with a thyro-iodine deficiency, i.e., is not a cretin, but *may be* related to a deficient pituitary secretion at the time pre-ossification changes are taking place.

Keith[22] believed the pituitary showed some histological abnormality ("especially a deficiency of those cells with large-cell bodies picked out by eosin"), but he adds that Professor Shattock thought the sections examined showed no pathological change. The pituitary gland is distinctly smaller in the achondroplasic than in the normal fœtus, but this may possibly be attributed to the defective growth of the basis cranii.

### THE AMNIOTIC PRESSURE THEORY.

The belief that achondroplasia will eventually be proved to depend in some way upon the internal glandular secretions is not universally accepted. In the past more than one author seems to have believed that pressure, exerted in different ways, would account for some of the phenomena; but its chief exponent is Dr. Murk Jansen, of Leyden. In an interesting and ingeniously reasoned monograph he finds "in the amnion the source of the deforming forces", and he considers the two important conditions to be: (1) Hydramnios; (2) Smallness of the amnion.

In hydramnios, a condition often present in achondroplasic fœtuses, the increased tension tends to make the amnion assume a spherical form, and so causes it to exert pressure on those portions of the embryo that project—namely, on the head, the neck, and the tail curves. As a consequence the embryo may become infolded, i.e., coiled up, and the pressure exerted by certain parts squeezed upon others results in deformations. For instance, the pressure on the neck and tail projections will account for the dorso-lumbar kyphosis often seen in these fœtuses. In other cases the amnion, from insufficiency of growth, may become too small, and when it completely surrounds the embryo, will again tend to infold it—the embryo accommodating itself to the small space by approaching the ball form. This infolding begins about the third week, when the embryonic tissues are very soft. But as the cartilage develops and the ossific centres form, the embryo becomes more resistant, and the amnion loses its power of infolding (six to seven weeks).

Besides the infolding process, the tension of the amniotic fluid is increased. In consequence of excessive secretion or want of growth of the amnion, ischæmia of the embryo must follow, for the vessels covering the exterior of the yolk sac are outside the amnion and the amniotic pressure squeezes the blood from the embryo into them. The diminished blood-supply produced in this way would interfere with growth, especially of those parts most recently formed and still soft—namely, the extremities. Ischæmia, in Jansen's[23] view, is the cause of the dwarf phenomena.*

* It is difficult to understand why proliferation of cartilage only should be deranged if the ischæmia affected the whole embryo and particularly the soft parts more than those slightly resistant. The development of the softer tissues might also be expected to suffer and to show some evidence of it in the fœtus or in cases that survive.

The theory of amniotic pressure is strongly opposed by Keith,[22] who voices the more generally held belief that "such indications as are available point to a hormone such as we may expect to arise either in the pituitary, thyroid, suprarenal or genital glands, or by an interaction of the secretion of all of them".

## THE SYNOSTOSIS OF THE BASIS CRANII.

In a disease in which the dominating feature is the feebleness of endochondral ossification, the fact that ossification at the base of the skull quickly obliterates the synchondroses calls for explanation (compare Mery and Labbé's case, p. 16).

There is no doubt that the synostosis takes place early. It is an important factor in the want of growth in the length of the base, though a certain share should be credited to deficient growth of the cartilage even before ossification commences. The ossifying process in the cartilage of the basis cranii is similar to that occurring in other cartilaginous parts of the achondroplasic skeleton. For fusion of the nuclei to occur so quickly the tracts of cartilage that separate them must be narrow, and their growth retarded. The cartilage of the spheno-occipital portion of the basis cranii is probably, therefore, unusually short to begin with, so that with the appearance of the nuclei only small synchondroses remain.

The original cartilage increases normally by the growth of cells from the perichondrium, and, when the ossific nuclei appear, by proliferation of its cells at the edge of the ossification zones. By the latter process the continued existence of the synchondroses is provided for. A deficient formation of cartilage by the perichondrium would account not only for the short spheno-occipital portion of the basis cranii, but for the want of lateral growth which is such a marked feature of this part of the base in the adult achondroplasic skull; and when this is combined with an absence of cartilage-cell proliferation, the narrow synchondroses, and the early fusion of the nuclei, can be understood. Clearly the lateral want of growth is not likely to be due to any fault in *periosteal ossification*, which in this affection is either normal or more active than usual in other parts of the skeleton. The mischief probably occurs before the perichondrium takes on the character and function of periosteum. A defective growth of cartilage, at any rate in the hypoplastic cases, is evidently an associated part of the cartilaginous lesion of achondroplasia, though it is usually thrown into the shade by the absence of the cartilage proliferation at the epiphysial lines, which is responsible for the short limbs.

## THE RELATION OF ACHONDROPLASIA TO CERTAIN BREEDS OF ANIMALS.

Souques, in his classification, has described a 'partial' form of the disease in which some of the usual characteristic signs are absent. Thus, in rare instances the achondroplasic conditions may be limited to the limbs, or even to the upper or lower limbs alone. It is the partial form which is supposed by many to account for the short bent limbs which distinguish certain peculiar breeds of animals. The 'bull-dog' calf is now generally recognized as an example of achondroplasia, but there has been a conflict of opinion about the

nature of the basset hound and the dachshund. In these animals the short distorted limbs associated with a long body are strongly suggestive of achondroplasia, but the skull is normal and shows no trace of the disorder. Moreover, their pups are born alive and possess the same peculiarities as their parents. Marie regarded them as perfectly normal beings when compared with the true achondroplasiac, who is always a pathological being, and in whom the contracted pelvis is a great obstacle to the propagation of the race. Regnault,[24] on the contrary, believed that they were the subjects of achondroplasia, and that an anomaly had been fixed intentionally by man to obtain, by means of selection, a race able to enter burrows and dislodge animals. He

FIG. 24.—The fore limb of a dachshund. (*R.C.S. Museum Stores.*)

FIG. 25.—The hind limb of a dachshund. The femur is slightly foreshortened. Note the position of the head of the fibula, which is still united by remains of its ligaments. (*R.C.S. Museum, Osteological Catalogue, Mammals*, No. 584A).

pointed out that nearly every breed of hunting dogs has its corresponding basset breed, and that in the time of Cuvier they distinguished between bassets with bent legs, and bassets with straight legs—the latter representing a slighter degree of the malady.

" Darwin records the case of a ram born in Massachusetts in 1790 having short crooked legs and a long back, which gave rise to an *otter* or *ancon* breed incapable of leaping " ; " in crossing with other sheep the offspring was always

perfect in resemblance to either parent, so that by selection a breed of this semi-monstrous character could have been very quickly originated".[25] The 'bull-dog' calf is born dead, but there is a race of cattle in Chile and Mexico called 'Natos' which have the bull-dog muzzle, and in some but not in all the limbs are very short. The front legs of a guinea-pig also are very short.

There is no doubt that achondroplasia shows a strong hereditary tendency, but if these peculiar breeds are really 'partial' forms of a diseased condition, not a perpetuated natural sport, the interesting point is that achondroplasia has become stabilized in a partial form. Though the question will remain unsettled until microscopical investigation decides the nature of the ossification process at the epiphysial lines, it may be pointed out that in the dachshund skeleton the head of the fibula is on a level with the head of the tibia (*Fig.* 25)—a definite achondroplasic peculiarity—whereas in the bull-dog it occupies a normal position (*R.C.S. Museum, Osteological Catalogue*, No. 589).

## TREATMENT.

In the present state of our knowledge of the pathogenesis of achondroplasia there is no sufficient indication for special treatment in those cases that survive, though cautious pituitary medication may be worth experimental investigation. But if a suitable treatment were known, there could be little hope of arresting the progress of an intra-uterine disease, which in the human being gives no inkling of its presence till the child is born.

In conclusion, I desire to express my gratitude to the many friends who have helped me in various ways in the preparation of this article, and especially to Sir Arthur Keith, Dr. Rodman, Dr. Greenfield, Mr. Plarr, and Col. M. H. Knaggs.

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# TRAUMATIC RUPTURE OF THE NORMAL SPLEEN.

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This paper deals exclusively with rupture by violence of the normal spleen. Thanks to its sheltered position behind the costal margin, this organ is comparatively rarely damaged. Having regard to the function of the spleen as a blood reservoir, and its variation in size, as registered by the oncometer, it is likely that a blow received just prior to a meal, when splenic congestion is at its height, would more probably result in rupture than at other times. But this interesting point must always remain a matter for conjecture.

Incidentally, doubt has been cast upon the prevailing opinion that splenic rupture is a commonplace occurrence in malarial[1] countries. McCracken[2] (1924) inquired of five of the most active surgeons practising in different sections of China (the home of the larang[3]), and found that they had not seen a single case of ruptured spleen, accidental or spontaneous. On the other hand, in the city of Shanghai rupture of the spleen is a fairly common industrial accident. Ruptured spleen appears, therefore, only to occur frequently where modern industry prevails.

The present paper is founded upon 32 cases, three of which have been under my own observation, and the remainder have been collected from the London Hospital records between 1894 and 1926. The spleen is evidently less frequently injured than the kidney. I was able to collect from the same records 135 cases of injury to the kidney[4] between the years 1902 and 1923.

**Sex Incidence.**—Of the 32 cases of ruptured spleen here reviewed, only 3 occurred in women over 14 years of age. The less arduous occupation, and the protection afforded by the wearing of corsets, doubtless accounts for this disparity.

**The History.**—There is the history of an accident. The nature of the violence was quite clear in every case in this review :—

| | |
|---|---|
| Street accident—run over or knocked down by a vehicle | 14 |
| Fall on to a projecting object, e.g., the corner of a table | 7 |
| Kicked in the abdomen | 3 |
| Fall from a height | 3 |
| 'Buffers' accident (compression) | 3 |
| Fall over handlebars of pedal cycle | 2 |
| | 32 |

Recently, Stretton[5] reported a case of ruptured spleen in which the history was most misleading. The patient, a woman of 37, stated that she was seized with violent upper abdominal pain whilst getting out of bed to pass urine. This author, not being satisfied with the history, made further inquiries, and found that the woman had been subjected to violent coitus two hours before the alleged onset of the symptoms.

*Loss of Consciousness.*—McCracken draws attention to the importance of a loss of consciousness immediately after the accident. A loss of consciousness

followed the injury in each of the three cases under my observation, and a careful perusal of the case-histories upon which the paper is based goes to show that an initial loss of consciousness is a frequent occurrence.

*Absence of Vomiting.*—In 11 instances it is recorded specifically that the patient had not vomited. Of the remainder, with the exception of 6 cases, there is no mention of it in the notes, nor is there any record of pre-operative vomiting in the patients' charts. I therefore feel satisfied that vomiting is somewhat exceptional in uncomplicated rupture of the spleen, and this point should prove useful when summing up data before making the diagnosis.

## GROUPING AND SYMPTOMATOLOGY.

Cases of rupture of the spleen may be conveniently divided into four groups: (*I*) *The patient rapidly succumbs, never rallying from the initial shock;* (*II*) *Initial shock—recovery from shock—signs of ruptured spleen;* (*III*) *The signs of an intra-abdominal disaster are delayed;* (*IV*) *Spontaneous recovery.*

### *Group I.*—RAPID SUCCUMBING OF PATIENT.

There are 3 cases belonging to this group. Complete avulsion of the spleen from its pedicle[6, 7] is the type of accident which is most likely to give rise to the symptoms which characterize this group.

A boy, age 5, was run over, and died two hours after admission to hospital. At necropsy the peritoneal cavity was found to be full of blood, and the detached spleen was discovered lying on the fundus of the bladder.

This is the only instance where the patient very rapidly succumbed. Fatal as it certainly is, if not surgically treated, uncomplicated rupture of the normal spleen cannot be classified fairly as a cause of *sudden* death.

### *Group II.*—SHOCK—SIGNS OF RUPTURE.

This is by far the largest group, and more than three-quarters of the total cases belong to it. After the initial shock has passed off, there are signs which point to a serious abdominal disaster. It is not always possible to state precisely which organ is damaged, but in the majority of instances the physical signs should point clearly to the spleen as the site of the injury.

General Signs.—It may here be emphasized how inconstant are the classical signs of internal hæmorrhage. I have repeatedly made notes upon patients in whom constant watchfulness has failed to reveal restlessness or air hunger, and yet at operation or post mortem they have proved to have a peritoneal cavity full of blood. In endeavouring to distinguish internal hæmorrhage from shock, it is surprising how insignificant is the help which can be derived from the clinical laboratory. For instance, one would think that a blood-count would help in the diagnosis of internal hæmorrhage; on the contrary, it is of little, if any, value. Even after a severe internal bleeding, all that this registers is a relative leucocytosis, and Brodin and St. Girons[8] have shown that there is also a leucocytosis in shock. Pallor is certainly a useful sign, but to wait for increasing pallor is often to court disaster. When the diagnosis is uncertain, a clearly recorded half-hourly pulse chart is above all things of the greatest diagnostic value. The general signs being unreliable, the local signs of ruptured spleen become increasingly important.

LOCAL SIGNS.—

1. *Abdominal rigidity* is variable, but it is present in more than half the total cases. It is often most pronounced over the left upper abdomen.

2. *Local tenderness* is very constantly found.

3. *Shifting dullness in the flanks* is probably regularly present. It was freely demonstrable in each of the three cases which I had the opportunity of examining personally. *Ballance's*[9] *sign* is said to be pathognomonic of splenic rupture: there is a dull note in both flanks, but on the right side it can be made to shift, whereas on the left it is constant. The interpretation of the sign is that there is blood in the peritoneal cavity, but the blood in the neighbourhood of the lacerated spleen has coagulated. There are many references to this sign in the literature, but the consensus of opinion appears to be that it so rarely present that it is almost valueless (Barnes,[10] Eliot,[11] etc.).

4. *Abdominal distention* commences to appear about three or four hours after the accident, and is probably due to ileus.

5. *Kehr's sign* is referred pain in the left shoulder.[12, 13, 14] There may be hyperæsthesia in this area. Though probably not especially looked for, left-sided shoulder pain was noted in three of the cases under review. Occasionally it is very much in evidence.

**A boy, age 5, fell off a wall. He walked home, but complained of abdominal pain. On examination in hospital late in the afternoon, there were very well-marked signs of ruptured spleen, but the boy's chief complaint was of pain in the left shoulder. The shoulder was examined, and nothing abnormal could be found, so the joint was X-rayed, but no abnormality was revealed. The spleen was successfully removed. There was no further note on the shoulder pain.**

MORTALITY.—Between 1894 and 1914 there were 11 cases, with 3 recoveries. Between 1914 and 1925 there were 12 cases and 11 recoveries; the case which died was complicated by a ruptured right kidney. These figures are a striking tribute to surgical progress.

### *Group III.*—THE DELAYED TYPE OF CASE.

Whereas all cases belonging to *Group II* had splenectomy performed between three and twenty-three hours after receiving injury, there remain 6 cases in which from three to thirteen *days* elapsed between accident and operation. In these 6 cases, which constitute *Group III*, after the initial shock had passed off the symptoms of a *serious* intra-abdominal catastrophe were postponed for a varying period. To quote an actual example which typifies this group:—

**A navvy, age 40, was hit in the upper abdomen by a pole. He fainted, but soon recovered sufficiently to walk to hospital, where he was examined and told to report the next day. On the morrow he felt better, and stayed at home. Five days later he was brought in with well-marked signs of internal hæmorrhage, having collapsed at home a few hours before admission. Splenectomy was successfully performed.**

Delay of serious bleeding may be explained in one of three ways: (1) The great omentum, performing its well-known constabulary duties, shuts off that portion of the general peritoneal cavity in the immediate vicinity of the spleen; (2) A bloody coagulum temporarily seals the rent; (3) A subcapsular hæmatoma forms, and later bursts. It is probable that each of these three factors, at one time or another, temporarily arrests serious hæmorrhage.

A point worthy of earnest consideration is the baffling friability of the splenic pedicle which is sometimes encountered. 'Friable pedicle' appears to be the peculiar terror of the delayed case, for it was found only in cases belonging to this category. The explanation of this phenomenon is fairly clear. The pedicle, after being surrounded by mildly infected blood and blood-clot for a varying time, itself becomes œdematous, and commences to undergo degeneration. It is thus more likely to be found in those cases in which serious hæmorrhage has been postponed by an omental barrier.

It behoves us therefore, when dealing with the delayed case, to take particular care to avoid this calamity. A mass ligature is more likely to cut out than a series of smaller individual ligatures applied by transfixion with a sewing needle close to the spleen. Further precaution is necessary to avoid 'losing' a pedicle which has cut out. C. H. Mayo has immortalized the slipped renal pedicle, which, he says, 'fairly jumps into the fingers' when nimble fingers promptly follow its retraction into the depths of the wound. If the lieno-renal ligament, with its vascular contents, slips or cuts through, it might be possible to retrieve it by the same means, but there are few of us who would not rather be forewarned!

MORTALITY.—Of the 6 cases belonging to this group, 3 died—two certainly, and one probably, directly owing to 'friable pedicle'. All the deaths occurred prior to 1914.

### *Group IV.*—SPONTANEOUS RECOVERY.

From time to time cases have been reported where the spleen was supposed to have been ruptured,[15,16] and with rest, and perhaps the administration of some ergot, the patient has recovered. Professor H. M. Turnbull informs us that neither he nor his colleagues have seen at necropsy a spleen containing the scar of an old injury. On the other hand, Sir Charles Gordon Watson[17] has described two specimens in the museum of St. Bartholomew's Hospital which contained *recent* scar tissue. The patients from whom these specimens were obtained died three and ten days respectively after an accident, from injuries other than those to the spleen. Inasmuch as no fewer than thirteen days have elapsed between the injury and the onset of serious symptoms in one of the cases included in *Group III* of this paper, who can say whether the scar tissue of the St. Bartholomew's specimens would have stemmed the tide of an improving blood-pressure had the patients lived? Again, hæmorrhagic solitary cysts of the spleen are generally attributed to injury. Whilst admitting the possibility, it is not clear why these cysts are usually found in women.[18]

The whole question of the natural repair of a splenic injury is an unimportant one. Nature fails so frequently that we must assume that surgical aid is always needed.

## OPERATION AND COMPLICATIONS.

**Operation.**—

*The Incision.*—The left paramedian incision is justly popular for splenectomy. Nevertheless, in an emergency the supra-umbilical mid-line incision

offers certain advantages. Foremost among these is the speed with which the abdomen may be opened or closed. In a doubtful case a useful practice is to make a 'button-hole' incision immediately above the umbilicus. If the diagnosis is confirmed, the incision can be rapidly enlarged towards the xiphisternum.

That the mid-line incision is usually adequate for removal of a ruptured spleen is, I think, well illustrated by one of my patients, a fat, barrel-chested man of 52, from whom the spleen was successfully removed by this route. Plumptre[19] has already drawn attention to the facility with which the splenic pedicle can be reached from this centrally-placed incision. In those rare instances where more room is required in order to deal with an adherent organ, the incision can be enlarged by a transverse cut to the left. Auvray's costal resection, which is recommended by Lejars[20] as the best approach for removal of a lacerated spleen, appears to be absolutely unnecessary.

*Dealing with the Injured Organ.*—Thirty-four years have passed since Riegues,[21] of Breslau, performed the first successful splenectomy for rupture. Since that time both tamponade and suture have had their advocates, but splenectomy has come into its own, and is now the standard operation for ruptured spleen. Splenectomy, in contrast to other methods, is rapid and certain. If time permits, it is advisable to examine the tail of the pancreas, and, if this is damaged, many complications may be minimized by a tube brought out on the left flank.

*The Question of Transfusion.*—An ideal method would be to transfuse the patient with matched blood as soon as the pedicle is ligatured. In practice there are many difficulties in the way. Even if a suitable donor is at hand, the necessary skilled assistance is not always available. Hauke[22] recommends transfusion of the blood from the peritoneal cavity. The blood is collected, mixed with citrate solution, and returned to the patient's veins. This collection of blood, especially as the incision is in the upper abdomen, must be time-consuming, but may possibly be facilitated by a suction apparatus. It must again be taken for granted that there is the necessary skilled assistance at hand, if this ingenious method is to be used.

In the majority of instances the infusion of saline must, of necessity, be substituted for the more ideal method of blood transfusion. A good method for the single-handed surgeon is to commence the operation by inserting the needles of a subcutaneous apparatus into the breasts, and allow saline to be absorbed during the operation. At the conclusion of the operation, if it is deemed necessary, the needles can be left *in situ*, and the infusion is continued when the patient is returned to bed.

**Early Complications.**—By early complications are meant those which occur during the patient's stay in hospital.

*Peritoneal Effusion*, amounting to ascites, was seen in one of my cases. It was noted on the eighth day after operation, and was accompanied by slight pyrexia. The fluid began to lessen in amount about the fourteenth day, but was demonstrable until the end of the fourth week. Its presence can be accounted for by a leakage of the pancreatic ferments from a wounded tail of the pancreas into the greater sac. It is well known that a large collection of fluid accumulates rapidly in the lesser sac after a traumatic lesion of the body of the pancreas.

*'Burst Abdomen'.*—The wound burst open, and had to be resutured under a general anæsthetic, in no fewer than four cases.* The most feasible explanation is that pancreatic ferments digest the edges of the abdominal wound and the catgut contained therein. Connors[23] noted three cases of severe suppuration of the abdominal wall following splenectomy for rupture. He reported that he had diligently searched the literature for the explanation, but had not found it. I submit that digestion of the edges of the wound, which undoubtedly occurs, would favour this infection. The tail of the pancreas may be wounded when splenectomy is being performed, or, as is probably more usual, it is injured together with the spleen at the time of the accident. There is good authority for the latter conjecture, for at the necropsy of one case of ruptured spleen which died without operation the tail of the pancreas was found to be almost severed.

*Left Pleural Effusion.*—A pleural effusion on the left side was noted in three instances, and was treated by repeated aspiration. The infrequency with which pleural effusion complicates fractured ribs makes it more probable that the effusion is due to bruising of the diaphragm at the time of the accident, or during removal of the spleen, than to a concomitant injury of the lateral thoracic wall.

*Persistent Hiccup.*—Hiccup was observed in several cases. A persistent hiccup, lasting more than five days, and preventing sleep, seriously complicated the convalescence of one patient; fortunately it was controlled by morphia, and he recovered. Hiccup is probably due to irritation of the branches of the left phrenic on the surface of the diaphragm.

*Splenic Asthenia.*—This occasionally complicates early convalescence.[24, 9] It commences about the tenth day after splenectomy. The patients become progressively emaciated. There is often some pyrexia. Irritability of temper is rather characteristic. The condition tends to improve on the exhibition of arsenic. The post-operative notes are not complete enough to draw any conclusions on the incidence of this train of symptoms.

The other complications—general peritonitis (two cases) and intestinal obstruction (two cases)—require no detailed consideration.

**Late Complications.**—The majority of the patients who have been traced report that they are in perfect health. The following information has accrued from tracing patients, and it throws some light upon the late complications of normal splenectomy.

*Attacks of Palpitation when Lying upon the Left Side.*—Two patients independently and spontaneously complained that for many months after their operation they were unable to lie in bed upon the left side without evoking an unpleasant attack of palpitation. Temporary cardiac embarrassment might result from loss of support to the under surface of the left cupola of the diaphragm. There is no denying that the spleen must offer some resistance to the excursions of the diaphragm in this region. The lack of this support, as far as the heart is concerned, would be most in evidence when the patient lay upon his left side.

*Fleeting Bone Pains.*—Pains in the bones 'like rheumatism' is a well-known late complication of splenectomy. It is due to the metamorphosis of

* The paramedian incision had been used in each of these instances.

yellow into red bone-marrow. There was only one instance in which this seriously worried the patient, and he stated that he was able to relieve the pain by rubbing his limbs.

*Attacks of Vomiting.*—During the first six months attacks of vomiting proved a considerable handicap to the return to normal life of three patients. The most striking instance was a patient who was employed as a flautist at a music hall. On returning to work he found that the act of blowing a flute caused him to vomit. This persisted for over three months, and then the symptoms abated. Possibly the loss of the spleen, a great blood reservoir, predisposes to passive congestion of the stomach.

Experiments on animals have led investigators[25] to believe that splenectomy lowers resistance to infection. It is often stated that, as far as man is concerned, there are not yet sufficient data upon which to settle this important question.[2, 16, 27, 28] I am able to trace 13 cases who have had a ruptured normal spleen removed. Two of these were operated upon 14 and 13 years ago respectively. In the remainder, between 3 and 11 years have elapsed since the operation. In no instance is there the slightest indication that a splenectomized person is more susceptible to infection than the rest of humanity.

I beg to record my thanks to the Surgeons of the London Hospital for permission to include in this paper cases under their care.

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# SPONTANEOUS RUPTURE OF THE SPLEEN.

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SPONTANEOUS rupture of the spleen is now a well-known catastrophe, but in most of the cases recorded the spleen was pathological; in many the disease had already been recognized; in others it was only found after operation or death. In the case reported here the spleen was apparently normal, and no history of injury was obtained from either the patient or his friends.

## SPONTANEOUS RUPTURE OF THE ABNORMAL SPLEEN.

This subject has often been written upon and discussed; it is briefly considered here as an introduction to spontaneous rupture of the normal spleen.

Malaria is the disease in which the spleen most frequently ruptures spontaneously.[1–6] Noland and Watson[7] found only 3 cases amongst 30,000 malarial patients at the Colon Hospital in eight years, but the condition seems to be commoner than these figures suggest. Of 123 spleens ruptured by injury, 93 were found by Berger to be malarial.

The condition occurs more rarely in typhoid fever. Connor and Downes[8] in 1914 could trace only 12 cases, in 9 of which it was found at autopsy; in the other 3, operation was performed for supposed intestinal perforation; none recovered. All were males, and the time of rupture varied from the second week to convalescence and relapse. Bryan[34] describes 25 cases, but in some of these the nature of the fever was doubtful.

The spleen has ruptured spontaneously in pregnancy, parturition, and the puerperium.[9, 10, 11] In these cases the spleen was softened and enlarged. Stretton[12] recently reported a case of rupture of a normal spleen in a woman five months pregnant; it seemed to happen spontaneously as the patient got out of bed, but Stretton thinks it really occurred during coitus a few hours before.

Hammesfahr[13] and Friesleben[14] have reported examples in leukæmia. A boy with hæmophilia has been cured of his disease by splenectomy done for spontaneous rupture[15]; this is claimed as the sixth case in which splenectomy has cured hæmophilia.

Rupture of the spleen may occur in the course of acute infections—for example, after appendicectomy and influenza[16]; with a carbuncle and marked toxæmia[17]; with a septic hand[18]; and with a whitlow[19]; in the last case there was a metastatic abscess in the spleen. It is rarer in tuberculous disease. Cannaday[20] had a case in which there was a spontaneous rent on the inner aspect of the spleen, which was studded with tubercles and occupied by caseous and calcareous deposits; the right kidney was also disorganized. Although this is an obvious example of rupture of a tuberculous spleen, it has been quoted in the literature as a case of spontaneous rupture of a normal

spleen.[21, 22] The remaining diseases in which the spleen has ruptured spontaneously are even rarer: typhus fever, relapsing fever, cystic degeneration, malignant growth, hydatid disease, infarction, torsion, abscess, and varices.

## SPONTANEOUS RUPTURE OF THE APPARENTLY NORMAL SPLEEN.

Connors,[21] writing in 1921, stated that he could find only two cases, those of Shorten[23] and Cannaday ;[20] but as the latter case was that of a tuberculous spleen it must be omitted. Moynihan[22] in 1926 wrote that in only three cases of spontaneous rupture was the spleen normal in size and in texture, but he also included Cannaday's case, while he omitted the two of Metcalfe and Fletcher.[24] I have found records of four definite and two doubtful cases.

**Connors' Case.**—Connors' patient[21] was a man, age 38, who had been treated for bronchitis and was suspected of having pulmonary tuberculosis ; he was a heavy drinker. Three months before admission to hospital he had complained of pain in the right hypochondrium, aggravated by deep breathing, and of nausea and constipation. On the day he came to hospital he had felt a severe pain in the left hypochondrium. Radiograms showed obstruction at the splenic flexure, and the pre-operative diagnosis was stricture of the colon, probably malignant.

At operation the gut was found to be normal. There was a hæmatoma beneath the capsule of the spleen, and the spleen itself was almost bisected longitudinally ; bleeding had ceased. Recovery followed splenectomy.

The spleen was apparently normal in size and consistency, but no microscopical examination was made. Subsequent inquiries negatived injury, malaria, or typhoid fever. The patient returned three months later, complaining of indigestion and abdominal pain ; there was a tumour in the left hypochondrium which was found at operation to be a cyst, probably pancreatic ; Connors considered that it was due to injury to the pancreas during removal of the spleen. This patient died of pulmonary tuberculosis four years later ; there is no record of an autopsy.

**Shorten's Case.**—This patient[23] was a man, age 43, who was admitted to hospital six hours after the sudden onset of peri-umbilical pain and vomiting. The abdomen was tender and rigid ; liver dullness was normal ; there was shifting dullness in the flanks. Signs of bronchitis were present.

At operation three hours later Shorten found fluid and clotted blood in the peritoneal cavity ; the spleen was almost divided into two equal parts, the tear extending from the convex surface to the hilum ; it was still bleeding. Splenectomy was done. The gall-bladder was full of small stones, but was not treated. The patient recovered. The spleen was normal in size and weight and histologically. There was no stripping of the capsule to suggest a preliminary subcapsular hæmorrhage, nor were there any adhesions about the spleen.

The patient's health had been good except for winter bronchitis. Eighteen months previously he had been buried in the trenches, whence he had been sent to hospital with severe abdominal pains ; he had been discharged after three months and had had no return of pain. In Shorten's opinion the absence of subcapsular hæmorrhage and of adhesions makes it unlikely that the injury received one and a half years before was in any way connected with the rupture.

The question of remote injury as a factor is discussed later.

**Metcalfe and Fletcher's Cases.**—These writers report two cases, both of which recovered after splenectomy.[24]

The first was a white male, age 21. He complained of sudden severe pain in the left side of the abdomen radiating to the left shoulder ; he vomited twice, and

was shocked. A few hours later he had paroxysmal pain in the left upper quadrant of the abdomen; he was tender here, but there was no rigidity or distension of the abdomen. The leucocyte count was 25,000 cells per c.mm. There was no history of injury or malaria; the Wassermann reaction was negative. The patient had had influenza and pneumonia a few years before. He had never had any gastric symptoms. At operation blood was found in the peritoneal cavity and the spleen was ruptured (the site is not stated). Splenectomy and recovery. The spleen appeared normal macroscopically and microscopically.

The second case was also a male white, age 21, who had had no serious illness or injury. After a large drink of cold water he was seized with cramp-like abdominal pain; he complained also of dizziness and pain in the left shoulder. He was admitted to hospital for observation; he got up next day, but again felt abdominal pain and dizziness; later the pain became worse and he showed signs of shock and internal hæmorrhage. There was no rigidity or distension (tenderness is not mentioned); the flanks were dull to percussion. The following day he seemed better, but still had pain in the abdomen and left shoulder; there was still no rigidity or distension, but the area of dullness in the flanks was larger. At operation blood was found in the peritoneal cavity and there was a clot around the spleen, which had ruptured on the convex surface. Recovery followed splenectomy. The spleen was normal macroscopically and microscopically.

**Skerritt's Case.**—Skerritt,[25] reporting his case of spontaneous rupture of the spleen, does not state definitely that the organ was normal, though his account indicates that it was. His patient was a man, age 53, who had been complaining of nausea, pyrosis, and anorexia for two months; once during this time he had severe epistaxis; he was admitted to hospital on account of a second attack of nose-bleeding. He was blanched; the radial pulse was feeble, and the vessel wall degenerate; a hæmic systolic bruit was heard. Nothing abnormal was found on abdominal examination. Later the patient became suddenly dyspnœic, and died in collapse within an hour; he had been lying quietly in bed for several hours before his death.

At autopsy much dark blood was found in the peritoneal cavity. The capsule of the spleen was distended with blood-clot, and there was a tear 1 in. long on the anterior part of the concave surface. The splenic substance was described as being soft and pulpy, due to considerable post-mortem changes. The spleen and clot together weighed 26 oz., but the weight of the spleen alone is not given. There is no report of a microscopical examination. The remaining organs were said to be anæmic but healthy.

**Atkinson's Case.**—This is the only case in which the patient was a female.[26] Her age was 35. She had not been well since her marriage five months before, complaining mainly of frequent attacks of vomiting and occasional epigastric pain. One night she became suddenly very ill, vomited frequently, and soon after had pain in the left hypochondrium, which radiated all over the abdomen. She died in a state of collapse twenty hours after the onset.

Post mortem a large clot weighing several pounds filled the left side of the abdomen; much fluid blood was also present. There were no adhesions, inflammatory lymph, or peritonitis. The spleen was described as being shrunken, pale, and flabby, and the lower pole as being in a state of muddy pulp.

Atkinson does not say whether he considered the organ to be diseased or not; no microscopical report is given. The other abdominal organs were healthy.

The last two cases were only very briefly reported, and must be considered as doubtful examples of spontaneous rupture of a *normal spleen.*

### Author's Case.

This patient was a man, age 53, a grocer by occupation; seventeen hours before admission to hospital he felt sudden severe pain in the upper part of the abdomen as he bent down to lift a bucket of water in his right hand. He had

retching, but was not sure if he vomited. His previous health had been good up to the last three months, during which he had had attacks of flatulence and indigestion after meals, but he had at no time been sufficiently ill to discontinue work or consult a doctor. He had never had any of the fevers, and the patient and his friends were quite sure that there had been no injury either recent or remote. Much of this information was only obtained after operation.

On Examination.—The patient was obviously very ill and in great pain; his temperature was 98·6°, the pulse was 80 per minute and of poor volume. He had well-marked arcus senilis. There was generalized board-like rigidity of the abdomen with marked distension; tenderness was most exquisite in the epigastrium and left hypochondrium. The abdomen was resonant all over and liver dullness greatly decreased. He had pain in the right shoulder as well as in the abdomen. A pre-operative diagnosis of perforated peptic ulcer was made.

Operation.—I opened the abdomen through a right paramedian incision. There was no free gas or fluid in this part of the peritoneal cavity; it was immediately obvious that the distension was due to gas in the transverse colon, which had to be punctured before the exploration could be satisfactorily continued. The stomach, duodenum, biliary tract, and appendix were all intact. The hand was then passed into the pelvis and brought out dripping blood; this was the first indication that there was any free fluid present. The spleen was soon found to be the source, and it was removed. There was no active bleeding at the time of operation and only 10 or 12 oz. of blood in the peritoneal cavity. The patient never improved, and died three days after operation with extreme paralytic ileus.

Autopsy.—Some clot was found about the pedicle of the spleen; the ligatures were intact and a small piece of pancreas was included in one of them. The whole of the gut was very distended. The other abdominal organs were healthy; so were the heart, aorta, and the splenic and renal vessels. The base of each lung was congested.

The spleen was normal in size, shape, and consistency; there was a hæmatoma, almost as big as the spleen itself, beneath the capsule on the convex surface, where there was a tear about 1½ in. long and ½ in. deep, transverse to the long axis and midway between the upper and lower poles. The spleen was carefully dissected in a search for some abnormality; but none was found.

Microscopical examination was made of the spleen, liver, kidneys, heart muscle, the splenic and renal arteries, and a lymph gland; all were histologically normal.

## CLINICAL FEATURES OF SPONTANEOUS RUPTURE OF THE SPLEEN.

It is generally agreed that these closely resemble those of traumatic rupture; but in the cases reviewed here there are some points of difference—for example, abdominal rigidity and tenderness, usually so marked in traumatic rupture, were not conspicuous features in the two cases reported by Metcalfe and Fletcher, nor in Connors' case; but they were well marked in Shorten's patient and in mine. Dullness in the flanks was found in the case of Connors and in one of the cases of Metcalfe and Fletcher; it was absent in my case and not mentioned in others. Ballance's sign, shifting dullness in the right flank and fixed dullness in the left, must cause distress, especially as it indicates a large amount of blood. Villalobos[27] has noticed a fine friction rub in the left flank, detectable by the stethoscope and by the hand.

Pain in the left shoulder has been noted in many cases of ruptured spleen, traumatic and spontaneous; it was present in both cases of Metcalfe and Fletcher. My patient said he had pain in the right shoulder, but he was mentally clouded when first seen and may have meant to say left; I can find no other case in which pain was referred to the right shoulder.

Trendelenburg,[28] discussing traumatic rupture, says that vomiting is a guide, as it is usually absent in cases of injury to the alimentary canal; on

the other hand, Melchior[29] states that vomiting is rare in rupture of the spleen. It occurred in Shorten's case, one of Metcalfe and Fletcher's, and in Atkinson's. Nausea was a symptom in Connors' patient, and my patient had retching.

Intestinal paresis was extreme in my patient, but was either absent or not obvious in the other cases. Nystrom[30] has reported three cases of ruptured spleen in which intestinal distension was severe; he added that he could find only one other case of ileus from this cause. In Nystrom's three patients the spleen was either sutured or tamponed, not removed; all recovered, but two required further operation for ileus. Hansell[31] has reported a similar case, and this may be the one to which Nystrom referred. In many cases unconsciousness has occurred; it is presumably syncope due to loss of blood; some of these were rapidly fatal without localizing features.

Does peritonitis ever occur? Hubbard[9] says: "Peritonitis has never been observed to follow rupture of the spleen, although it follows rupture of the liver, pancreas, and other abdominal organs". Ledderhose[32] writes that "After rupture of the spleen, peritonitis does not occur". Gordon-Watson,[33] however, states that "If the patient survive the hæmorrhage and no operation be performed, he may yet succumb to peritonitis". Moynihan[22] mentions peritonitis as a possibility in unoperated cases. Gordon-Watson and Moynihan do not state if they have ever found peritonitis present at operation; they only mention it as a possibility in unoperated cases, so it must be a late event if it does occur. There was no peritonitis in my patient at the operation or at autopsy, and it is not recorded in any of the other cases reviewed here.

In my patient there was no more than 10 or 12 oz. of blood in the peritoneal cavity, his poor condition being due more to paralytic ileus than hæmorrhage; I found blood only on exploring the pelvis and left paracolic region. Melchior[29] has drawn attention to the point that in ruptured spleens the extravasated blood tends to spread at first only in the left half of the abdomen, whence it reaches the pelvis, and only when this is filled does the blood rise into the right half of the belly. Bryan,[34] in a patient with a ruptured typhoidal spleen, only found blood in the pelvis and during evisceration.

**Site of Rupture.**—Of these 7 cases in which the spleen was apparently normal, rupture was on the convex surface in 3 (Shorten's, one of Metcalfe and Fletcher's, and mine); in 1 (Skerritt's) it was on the concave side; the site of rupture in the remaining three is not stated.

Cantlie[35] calls attention to the frequency of rupture on the concave surface in malarial spleens resulting from trauma, although in many cases of spontaneous rupture it is on the convex side. Leighton,[36] reporting a case of rupture on the convex side of a malarial spleen, quotes Deadrick,[43] who says that laceration occurs with equal frequency on the concave and convex sides.

Friesleben[14] states dogmatically that "the rent in spontaneous ruptures, in contrast to traumatic ones, is always on the convex side", but in Skerritt's case the tear was on the concave side.

Connor and Downes,[8] writing on rupture of the typhoidal spleen, say that the rupture may involve any part of the surface and is usually linear. Ledderhose[32] also states that the rupture may be situated on any part of the

organ; multiple rents have been found, and in rare cases complete separation of the spleen into two or more pieces has occurred.

## MECHANISM AND ETIOLOGY OF THE RUPTURE.

The mechanism of rupture of a pathological spleen, especially the malarial and typhoidal, has been discussed by many writers: Bryan,[34] Nolan and Watson,[7] Ledderhose,[32] Friesleben,[14] Foucault.[37] All have closely identical views on the causes, which may be summed up as: (1) Softening of all the structures of the spleen; (2) Congestion of the portal vein and its radicles, and inability of the narrow splenic vein to accommodate itself; (3) Blood is thus forced between the spleen and the investing peritoneum, and finally the latter gives way; (4) Perisplenic adhesions, by fixing the organ, predispose to rupture.

With many ruptured spleens, as in my case, a subcapsular hæmatoma has been formed; this supports the suggestion that there are two stages, first an effusion beneath the capsule, then secondary rupture of the latter. A similar process is known to occur in some cases of rupture of the liver. These subcapsular hæmatomata may form cysts, and several cases of successful operation at this stage have been reported (Planson[38]).

Wohl[39] points out that histologically the spleen ages early, and he thinks that these early degenerative changes predispose to its rupture; at the age of 36 nearly 50 per cent of spleens show thickening and hyaline changes in the Malpighian bodies and capsule, and the blood-vessels become thickened. But these factors must be subsidiary, otherwise it would be more common and would increase in frequency with age, as do, for instance, rupture of the fibroid heart and spontaneous fracture in degenerate bone—the neck of the femur, for example.

Ledderhose[32] and Foucault[37] are both sure that spontaneous rupture occurs exclusively in the diseased spleen. If they are correct, then in the cases here discussed the spleen is only apparently healthy; yet no evidence of disease was found. A possible explanation is that the spleen is abnormal at one area only, that rupture occurs at this spot, and all evidence of pathological change is destroyed by the disintegration associated with the rupture. Of the seven cases, five had digestive symptoms; two of these had conditions in other organs sufficient to account for these symptoms—namely, gall-stones and pulmonary tuberculosis respectively; in the remaining three no cause for the gastric symptoms was discovered. I suggest that some localized disease in the spleen caused these symptoms first, and later the spontaneous rupture which obliterated evidence of the disease. My patient had complained of flatulence and indigestion for three months; Skerritt's patient had had nausea, pyrosis, and anorexia for two months; and Atkinson's had suffered from occasional epigastric pain and frequent vomiting for five months. This suggestion receives further support from the fact that the digestive power of the gastric juice is constantly weakened after removal of the spleen; it is conceivable that a gradual loss of the spleen by disease would similarly affect the gastric juice; it is supposed that the spleen provides a substance which causes the gastric glands to secrete more pepsin.[40] The close anatomical relation of the spleen to the stomach, and the rhythmical contractions which

it undergoes during digestion, still further make it likely that early splenic changes produce gastric symptoms. Finally, well-known splenic diseases such as Banti's disease are frequently mistaken at first for gastric or duodenal disorders.

Of the seven cases, only two, those of Metcalfe and Fletcher, had no gastric symptoms; they were both vigorous young men of 21 and may have had minor symptoms which were not mentioned.

In none of these cases was there any evidence of softening and congestion, the conditions which are blamed for spontaneous rupture of enlarged spleens, as in malaria and typhoid fever. There remains the possibility of some remote injury, perhaps forgotten or unnoticed, being the underlying cause of the rupture. Remote injury was considered in the case of Shorten, but was negatived for the reasons given; in each of the remaining cases injury is definitely denied. But a case reported by Jackson[41] warns us that rupture of the spleen may occur at an appreciable time after an injury and the association between them be missed.

**Jackson's Case.**—The patient was a girl, age 15, who suddenly developed pain in the left lower quadrant of the abdomen while at a cinema show; she vomited, and showed signs of collapse; the belly was rigid all over, and there was dullness in the flanks. On asking about her previous health one only learnt that she had complained of not feeling well for several days about a month before.

At operation blood was found in the peritoneal cavity; the source was a rent of the convex surface of the spleen, which was large and œdematous. Recovery followed splenectomy. It was only during convalescence that the child was able to give a reliable history; she stated then that she had hurt her left side against a desk at school twenty-eight days before the operation; she complained only of not feeling well and did not tell anyone of this injury. This case might easily have been counted as one of spontaneous rupture if the patient had not lived to give her history.

**Lemprière's Case.**—This author[42] had a male patient, age 15½ years, who was injured at football; he had epigastric tenderness, vomiting, and shock, but seemed to recover completely in a few days; on the eighth day he played squash rackets and suddenly collapsed with acute abdominal pain. At operation a rupture was found on the concave surface of the spleen. The interval of eight days between the injury and the rupture might well have been prolonged if the boy had rested longer.

Two specimens in the Museum at St. Bartholomew's Hospital support the possibility suggested by these two cases, that temporary, if not permanent, healing of a ruptured spleen may occur. These specimens are described by Gordon-Watson.[33] One is the spleen of a woman, age 30, who fell thirty feet and fractured a femur; she died ten days later without ever having abdominal symptoms; there was blood about the spleen, which had evidently been torn across, and a firm white scar had formed between the lacerated surfaces. The other specimen was removed after death from a woman, age 39, who was run over by an omnibus and died sixty hours later. The spleen had been torn on the outer surface near the upper pole and the rent was closed by a firm clot.

There must be other similar cases and specimens. These few are cited to show that an apparently spontaneous rupture of the spleen may really be a traumatic one with delayed symptoms—due either to the bleeding being subcapsular at first and intraperitoneal later, or to a temporary clot in the tear yielding after an appreciable interval.

**SUMMARY.**

1. A case of spontaneous rupture of an apparently normal spleen is recorded; only six similar cases could be found in the literature.

2. Of the seven cases, five had previous digestive symptoms; of these five, one had gall-stones and another pulmonary tuberculosis and alcoholism; in the remaining three no cause of the digestive symptoms was found; possibly they were due to some splenic lesion, evidence of which was destroyed at the time of rupture.

3. Injury to the spleen may cause no immediately alarming symptoms, so that the association between an injury and subsequent rupture of the spleen is overlooked. Post-mortem specimens of healed spleens, and clinical cases of delayed rupture of the spleen, have been described to show that some cases of apparently spontaneous rupture are really traumatic.

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# CANCER OF THE TONGUE: PRELIMINARY REPORT ON RADIUM TREATMENT.

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Impressed by the results obtained by Professor Regaud at the Institut de Radium in Paris, we have given his methods due trial in cases of lingual cancer. The results have been so gratifying that we consider them worthy of record. For the present we make no comment on the theories underlying radium therapy or on the physical or biological problems; we are here concerned only with the clinical findings in seventeen cases of undoubted carcinomata of the tongue and of the changes effected in them by the application of radium. In each case the growth treated was examined microscopically; the local appearance at intervals can be seen in Mr. Sewell's drawings; the changes detected by palpation are noted. The method of radium application and the dosage in each case are given.

That the changes effected locally are striking is obvious; in some cases the effect on malignant structures somewhat remote from the site of the primary lesion is also striking (*see Figs.* 38, 39). As to whether these changes are permanent, time alone will decide, but the disappearance of the neoplasm appears to be so complete that in our opinion even early and operable cases should be submitted to radium rather than any other form of treatment, including surgical excision and diathermy.

The problem presented by a case of lingual cancer is twofold: the planning of the actual radium therapy of the primary growth; and the treatment of the lymphatic area draining the tongue.

## CONDITIONS AFFECTING TREATMENT.

**Type of the Growth.**—We are only considering the squamous-celled carcinomata. As a rule it can be stated that the condition of the surrounding area profoundly affects the results obtained. Cases where the growth is of the fungating cauliflower type or of the smaller papillary form give good results. The ulcerative type is rather more resistant to treatment, but if small gives equally good results. The worst type of growth is that which is embedded in an œdematous zone—whether the growth be of the nodular infiltrating variety or of the fissured ulcer type.

Whatever the karyokinetic activity of the malignant cells may be, they respond to irradiation; but if the stroma be œdematous, it is not sufficiently resistant to the irradiation however well screened the radium may be; its blood-supply is interfered with, and radionecrosis is very probable (*see Case* 2).

The suitability of a case for radium therapy depends upon two principal factors: (1) The degree of extension of the lesion; (2) The anatomical

position of the lesion. The question of glandular involvement does not *per se* materially alter the treatment of the primary growth, although it profoundly influences the prognosis and demands a more careful consideration of dosage. Even with obvious glandular infection the primary growth can be got rid of, and then the question of the treatment of the lymphatic area is considered separately.

**Size of the Growth.**—The smaller the growth, the easier the treatment and the better the prognosis. A large carcinoma presents difficulties of adequate protection of the surrounding bony structures, as a larger quantity of radium is required; hence the greater danger of radionecrosis.

**Anatomical Position of the Growth.**—Epitheliomata of the tip, lateral border, and dorsal aspect of the tongue anterior to the V-shaped row of papillæ are most suitably situated for treatment by radium, since they are easily accessible and the jaws can be efficiently protected by a simple apparatus containing a lead lining. The apparatus interferes very little with deglutition, it can easily be kept clean, and the pathological area is under continuous supervision. Epitheliomata of the inferior surface of the tongue are harder to treat, and when the floor of the mouth is invaded by the disease the difficulty is still greater, for it has now become less easy to design an apparatus which will efficiently shield the lower jaw from necrosis. But even the fear of necrosis should not deter from the use of radium, seeing that surgical treatment of the same condition will necessitate excision of part of the floor of the mouth, and possibly of the adjacent portion of the mandible.

Epitheliomata of the posterodorsal part of the tongue and of the posterolateral part in the vicinity of the pillars of the fauces are the most troublesome to deal with, not only on account of the difficulty in gaining access to the tumour, but also because the proximity of the growth to the angle of the jaw and the palate makes it necessary to protect these structures from necrosis, and efficient protection is not only hard to maintain, but must increase the already existing discomfort of the patient. Since these growths tend to spread to the pharyngeal portion of the tongue, the most careful examination must be made lest the extent of the disease be underestimated.

## TECHNIQUE.

**Preparation.**—As a preliminary to radium therapy the most scrupulous toilet of the mouth is essential. All carious teeth are extracted; tartar is scraped; gold bridges or large metal fillings must be sacrificed; frequent mouth-washes are employed till the gums are healed and infection is eliminated as far as possible.

It is our custom to treat *the primary growth* first, and then *the glands*: they are never treated at the same time.

### 1. THE PRIMARY GROWTH.

Both lingual nerves are injected with 1 per cent novocain, and if necessary both inferior dental nerves are also anæsthetized. As a rule no general anæsthetic is employed. The growth is examined, and its situation, extent,

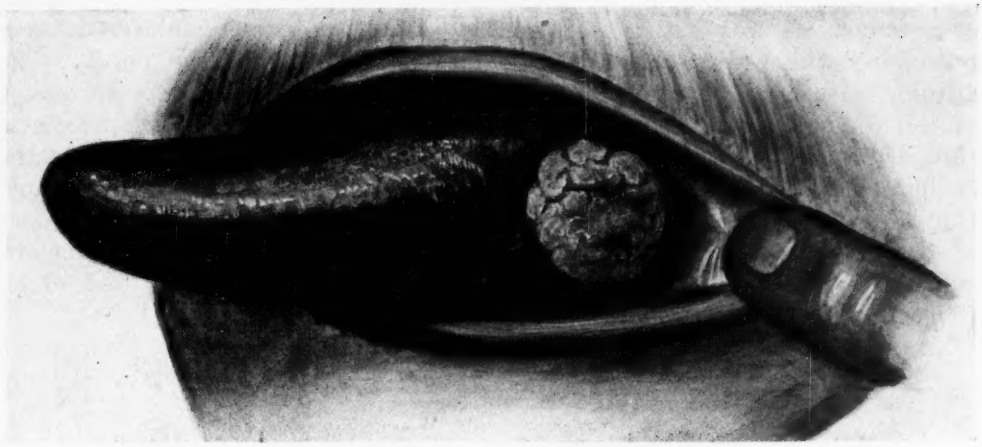

FIG. 26.—*Case* 1. Before treatment.

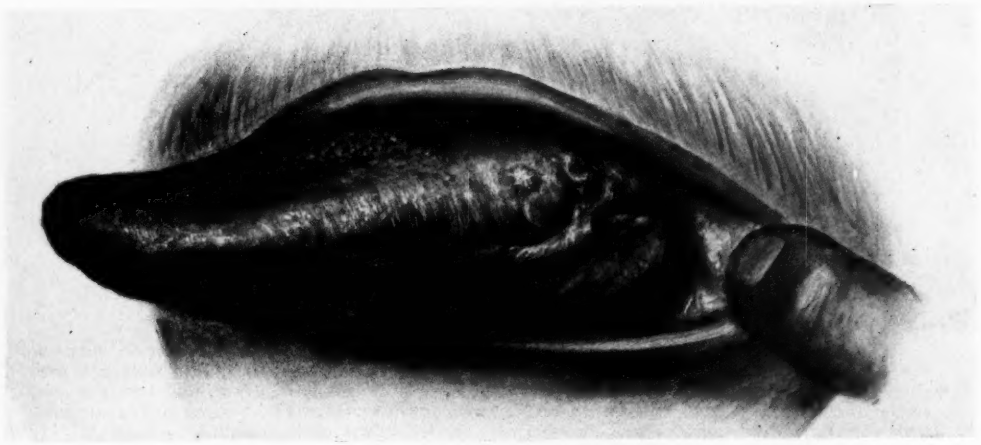

FIG. 27.—*Case* 1. Condition two months after treatment.

FIG. 28.—*Case* 1. Condition fifteen months after treatment.

and 'volume' are ascertained. The depth of the growth is estimated as far as possible. A stay suture is introduced into the tongue and serves as a retractor; the assistant holds this and he also retracts the cheek. The growth is surrounded by needles which are introduced into the substance of the tongue in the immediate vicinity of the neoplasm. Each needle is threaded with a silk suture and with a piece of fine silver wire. When the radium has been buried, the silk suture threaded on a needle is passed through the substance of the tongue, tied, and cut short. The silver wire protrudes at the angle of the mouth; all the wires are threaded through a narrow rubber tube to protect the lips, and the tube is strapped to the cheek (*Fig.* 29).

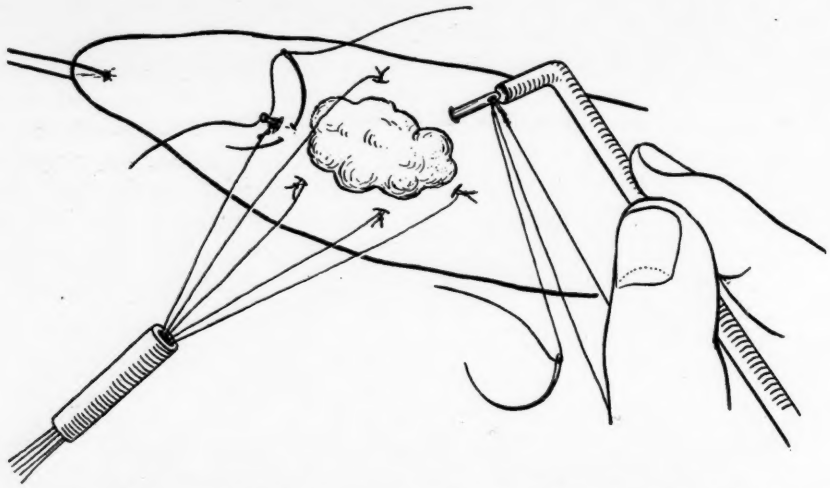

FIG. 29.—Method of introduction of the radium needles. The silk retention sutures are also shown. The silver wires, after the introduction of all the radium needles, are threaded through a rubber tube, longer than that shown in the diagram, and brought out at the angle of the mouth; they are then strapped to the cheek. These wires make it impossible for the radium needles to be swallowed, and facilitate their removal.

**Dosage.**—Each needle contains radium bromide equivalent to 0·6 mgrm. of radium element; it is 16 mm. long, has a diameter of 1·65 mm. and is screened by 0·5 mm. of platinum. Between 6 and 10 needles are used according to the size of the growth; they are introduced at equal distances from each other into healthy tissue in the immediate vicinity of the growth (*Fig.* 29). They are left in position from 6 to 8 days, the total dosage varying from 600 to 1100 mgrm. hours. This method of calculating is employed as we never use emanations. The points noted are: the number of needles, their size, strength, screenage, and the duration of application. To protect the adjacent bone a simple apparatus is made containing a sheet of lead 1 mm. thick, covered with vulcanite or stent, moulded over the jaw and teeth. This is made specially for each individual case, and the shape and extent of lead protection vary with the position of the growth. For growths situated far back it may be necessary at times to resect subperiosteally the angle and part of the ascending ramus of the mandible, as advocated by L. Ledoux, of Brussels.[1] This resection is done under regional

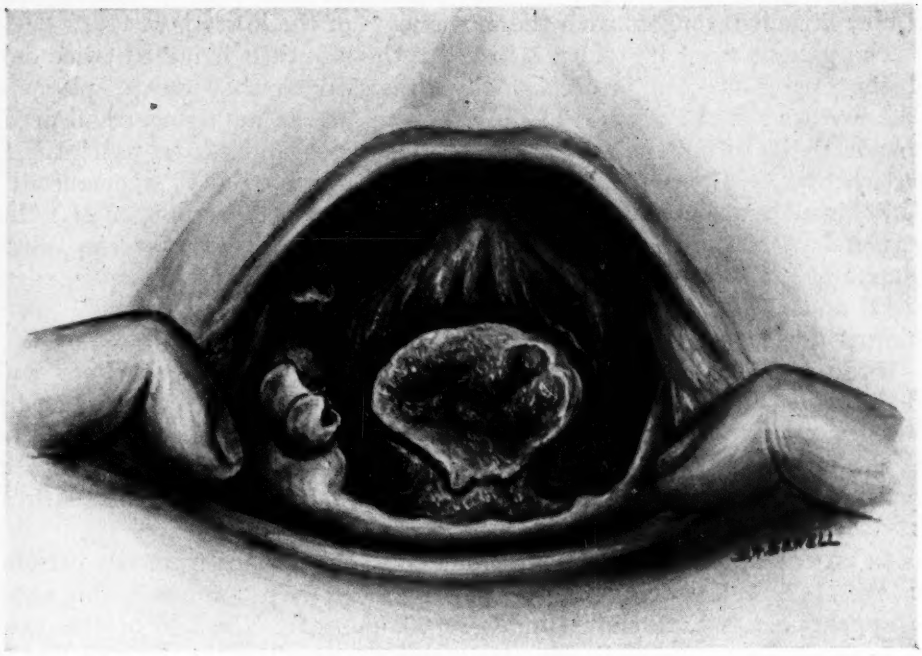

FIG. 30.—*Case* 2. Before treatment.

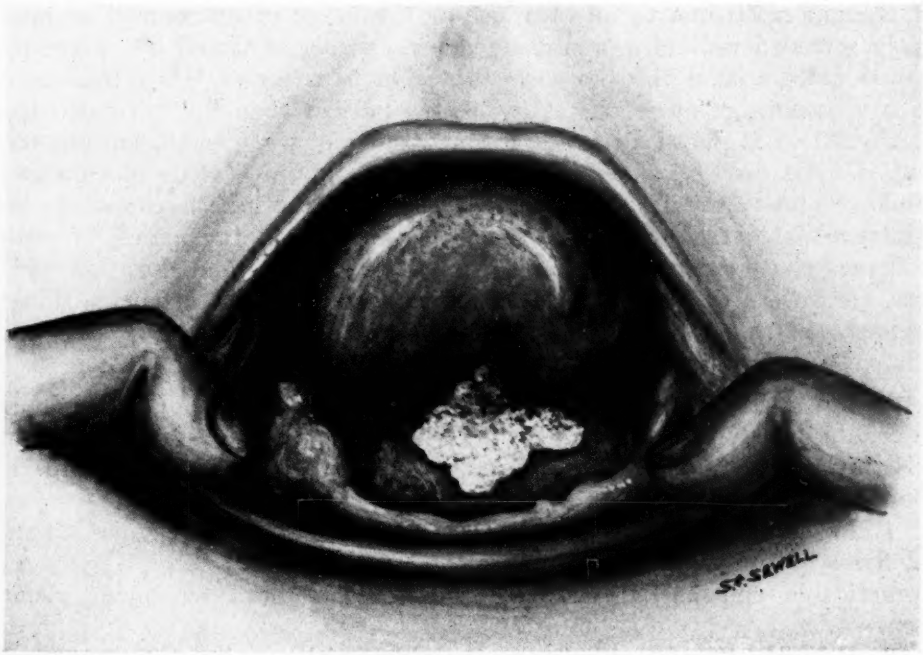

FIG. 31.—*Case* 2. Showing œdema of tongue and slough due to radium necrosis two and a half months after treatment.

anæsthesia. It is surprising how little discomfort is caused by this operation and how little it interferes with the movements of the lower jaw.

The patient must be told not to talk; the mouth is irrigated twice daily, and the operation area is occasionally inspected so that any displacement of the needles may be remedied. The introduction of numerous small needles demands patience both on the part of the surgeon and of the patient. The after-treatment is extremely important; the nursing must be entrusted to an intelligent and capable nurse who has been taught the danger of radium burns and of radionecrosis, and the patient must be encouraged to tolerate the apparatus in the mouth. With careful selection of suitable cases, correct dosage, and the right screenage, the disappearance of the primary growth is almost assured.

**Mode of Disappearance of the Growth.**—The tumour becomes softer and the surface of the growth takes on a whitish appearance, as if it had been painted with pure carbolic acid (*see Fig.* 41). The growth becomes flattened, and the induration which before was one of its striking characteristics disappears. *Figs.* 36 and 37 show the changes in appearance on the tenth and thirty-seventh days.

In an ulcerated neoplasm epithelialization takes place from the periphery (*see Figs.* 42, 43), leaving hardly any scarring. On removing the needles the appearance is that shown in *Figs.* 36 and 41. The site of the needle punctures is seen as small necrotic points, and the area treated appears inflamed. The result in successful cases is such that it is difficult within three months to find the site of the original growth (*see Figs.* 37, 28, 39), and the mobility of the tongue returns to normal.

Radionecrosis due to an overdose or the usage of unscreened or insufficiently screened radium is a prolonged process, accompanied by severe pain, marked œdema, and the presence of a slough extremely slow to separate, which is sometimes more exhausting to the patient than the untreated lesion (*see Fig.* 31). It may lead to the disappearance of the growth, but the treatment is most distressing. Between these two extremes of disappearance by shrinkage and epithelialization and that by necrosis there is the whole scale of intermediate stages. With increased knowledge of the action of radium on tissues the optimum dosage in amount, screenage, and duration will be more accurately defined, and radionecrosis should then become a thing of the past.

### 2. GLANDULAR INVOLVEMENT.

With regard to the treatment of the glandular area, the difficulty consists in deciding which cases are best treated by excision, which by radium therapy, and which by a combination of both. Clinically these cases fall into three groups :—

1. *Frankly Inoperable Cases.*—The glandular involvement is extensive and fixed to the main vascular sheath. As a complete surgical removal is impracticable and partial removal inadvisable, radium treatment alone is given.

2. *Palpable Glands.*—Clinically operable, with no involvement of subcutaneous tissues. Surgical resection is done. It is preferable to do a complete resection on both sides of the neck.

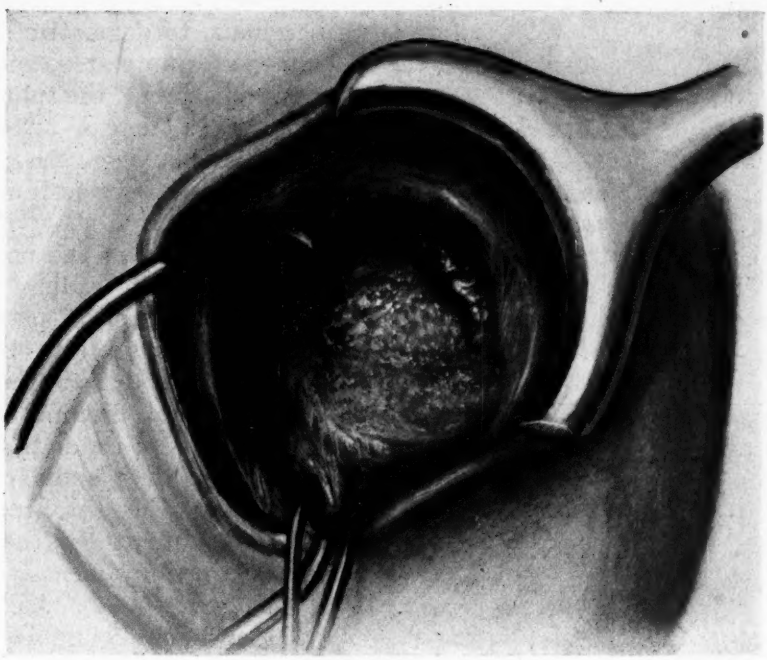

FIG. 32.—*Case* 3. Before treatment. Hemiglossectomy had been done twelve years previously. Recurrence on the right side.

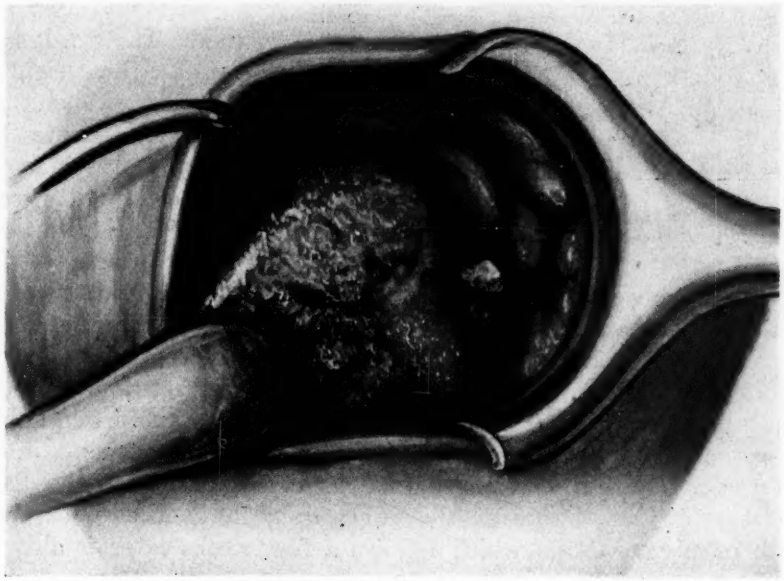

FIG. 33.—*Case* 3. Condition two months after treatment.

3. *No Palpable Lymphatic Invasion.*—It is in the treatment of cases falling into this group that we are faced with the greatest difficulty. Our knowledge is at present too incomplete to permit us to dogmatize as to the safest method of treatment. We have left some cases untreated, and several months later, though the tongue was normal in appearance, the submaxillary and carotid glands became enlarged (*Cases* 1 and 4), in one case appearing on the side of the neck opposite to that of the primary growth (*Case* 6). Prophylactic irradiation of the neck may prevent this late appearance of the lymphatic involvement, but further experience alone can show if this is preferable to systematic glandular resection of both sides of the neck.

**Treatment.**—The treatment of lymphatic infection by radium is made entirely by means of distance application. The introduction of needles into the tissues of the neck has been discarded owing to the bad results obtained—these are due to the impossibility of distributing equal amounts of irradiation in the area of the glandular enlargement. The distance between the radium and the skin is obtained by means of plastic collars (*Fig.* 34). A compound known as Columbia paste is used for this purpose; it was described by O. Monod, A. Esguerra, and G. Richard.[2] It consists of beeswax, 100 grm.; paraffin (melting at 62° C.), 100 grm.; and fine sawdust, 20 grm. The sawdust greatly diminishes the weight of the apparatus. When properly prepared the paste has one yellow smooth surface and one dark-brown slightly granulated surface. It is important to have the paste of uniform thickness; 15 mm. is a useful working thickness. As the paste contains no metallic substance, no secondary β rays are to be feared. Further, it has been proved at the Radium Institute in Paris that this medium produces a homogeneous irradiation and a diffuse superficial distribution of the rays.[2] This is demonstrated by the fact that when the apparatus is removed at the end of the treatment, if a dose sufficient to produce an erythema of the skin has been given, the erythema is situated, not corresponding to the disposition of the several needles, but over the exact area in contact with the paste. The patch of pigmentation which follows a few weeks later is also the size and outline of the paste.

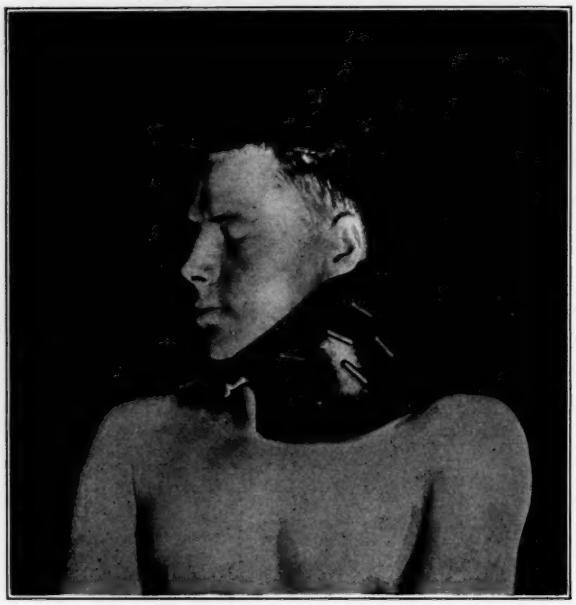

FIG. 34.—Columbia paste collar with radium needles in brass capsules.

Heated at 48° C. the paste becomes soft and malleable but not brittle; it can be handled with ease and suitable pieces cut out; these are accurately

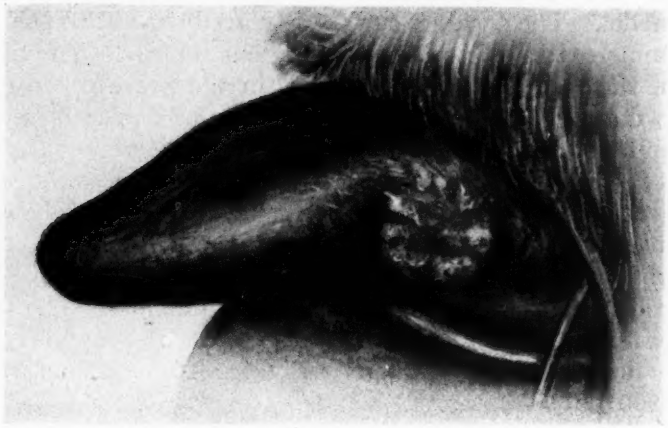

FIG. 35.—*Case* 4. Before treatment. The patient also had an epithelioma of the pillar of the fauces, shown in *Fig.* 38.

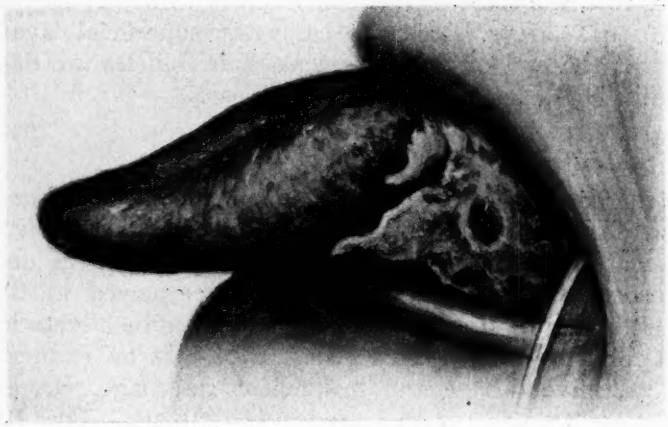

FIG. 36.—*Case* 4. Condition on the tenth day.

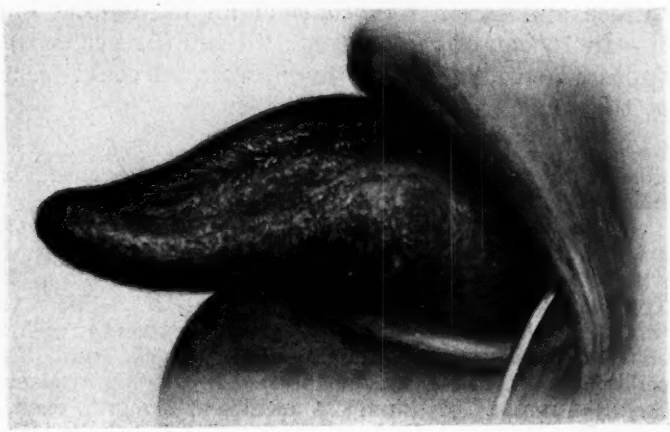

FIG. 37.—*Case* 4. Condition five weeks after treatment.

moulded over the neck either directly or on to a previously prepared plaster model. The sawdust surface is placed nearest to the skin. The model is then immersed in cold water and hardens rapidly. The next step is the disposition of the radium tubes or needles in the previously prepared wax collar. This is done by gently heating each tube or needle by passing it through a flame and applying it on the external, smooth yellow surface of the apparatus.

For the prophylactic treatment of the neck in cases where no glands are palpable, we use 40 mgrm. of radium equally distributed over the submaxillary and carotid areas. By employing 20 needles or tubes of 2 mgrm. of radium element each, screened by 0·65 mm. of platinum and evenly distributed, a uniform irradiation is obtained. The thickness of 15 mm. of paste acts as a further protection. The application is intermittent, the patient receiving 8 hours daily for 10 to 14 days, the total dosage being 3200 to 5400 mgrm. hours.

In cases where the glands are greatly enlarged and inoperable, it is necessary to employ bigger doses—between 60 and 80 mgrm ; a double thickness of paste (30 mm.) is used ; the application is continuous day and night, with a few brief periods of rest, the total dosage being 14,000 to 25,000 mgrm. hours distributed over 10 to 14 days. In these cases the superficial layers of the skin peel off towards the end of the treatment, hair follicles are destroyed, but the skin readily heals, leaving a pigmented scar.

## RECORD OF CASES.

The following seventeen cases were all examined histologically and found to be squamous-celled carcinomata. The primary lesion was treated with radium only ; the treatment of the cervical glands varied in each case, and the particular method chosen is indicated. The dosages employed in the first three cases were much bigger than what we now consider to be advisable, and were left in position for a short period of time. *Case* 2 is an example of radionecrosis due to excessive dosage and insufficient screenage. In all cases except *Case* 12 and *Case* 3 needles were buried in the tongue. *Case* 12, owing to previous syphilitic infection and the invasion of the whole tongue by the neoplasm, was unsuitable for radium puncture, and was treated by surface application by means of an intrabuccal apparatus.

In 16 out of the total of 17 cases (94 per cent) the primary growth disappeared entirely and nothing abnormal could be detected in the substance of the tongue on palpation.

*Case* 1.—Mr. A. C. G., age 62. Nodular growth the size of a shilling on the left side of the tongue (*Fig.* 26) ; no palpable cervical glands. Biopsy : squamous-celled carcinoma.

TREATMENT.—The following amount of radium was inserted : 2 needles at 10 mgrm., 2 needles at 12·5 mgrm., and 1 needle at 25 mgrm. All needles were screened with 0·5 mm. of platinum and left in position 12 hours. Total dose 840 mgrm. hours. The growth disappeared entirely (*Figs.* 27, 28). Six months later an enlarged gland was excised from the left anterior triangle of the neck and proved to be epitheliomatous on histological examination. A prophylactic irradiation with a Columbia paste collar 30 mm. thick carrying 60 mgrm. of radium element (20 needles of 2 mgrm. each, 0·6 mm. of platinum ; 4 needles at 5 mgrm. each, 0·5 mm. of platinum) was applied 10 hours daily for 7 days. Total dose 4200 mgrm. hours. The patient remains well up to the time of writing (two years).

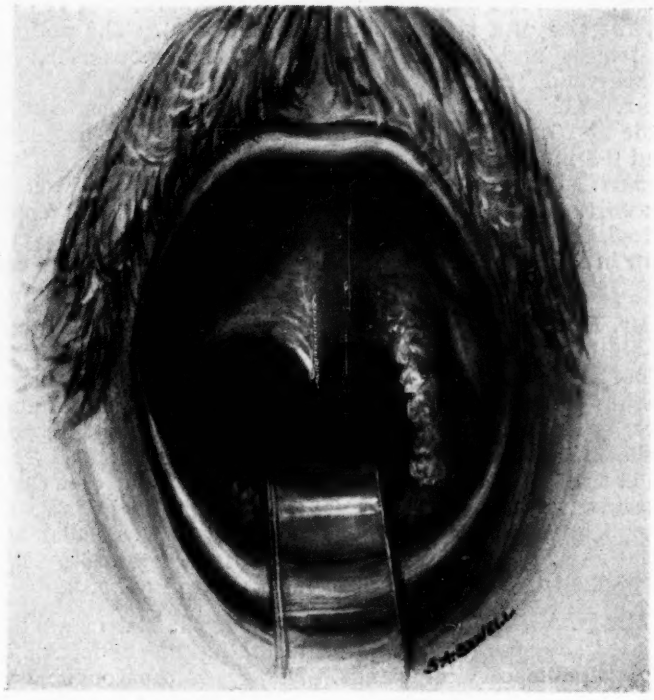

FIG. 38.—*Case* 4. Epithelioma of pillar of fauces before treatment. Patient also had an epithelioma of the tongue (*Fig.* 35).

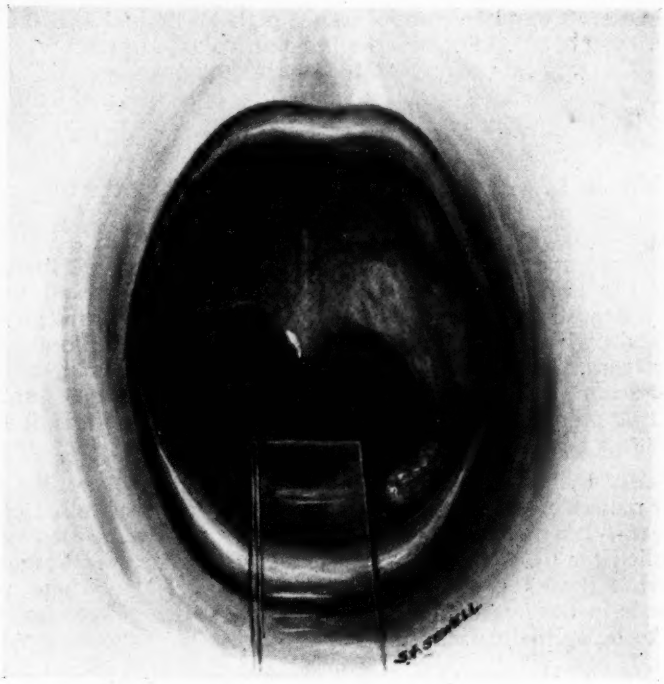

FIG. 39.—*Case* 4. Three weeks after treatment.

*Case* 2.—Mr. T. C., age 54. Ulcer on the under surface of the tongue and floor of the mouth, the size of a two-shilling piece (*Fig.* 30). The patient was suffering from mitral stenosis and glycosuria. Biopsy: squamous-celled carcinoma. No enlarged cervical glands palpable.

TREATMENT.—The following amount of radium was inserted: 1 needle at 50 mgrm., 1 needle at 25 mgrm., 2 needles at 12·5 mgrm., and 5 needles at 10 mgrm. All needles had 0·5 mm. of platinum screenage. Application 14 hours. Total dose 2100 mgrm. hours. Radionecrosis resulted: marked œdema of tongue, severe pain. extreme discomfort. The growth entirely disappeared and was replaced by a white slough which took three months to separate. The condition at the end of the second month is shown in *Fig.* 31. The patient died of exhaustion at the end of the fourth month.

*Case* 3.—Mr. E. S., age 68. There had been right hemiglossectomy twelve years previously for epithelioma of the tongue. Recurrence took place in the scar, involving tongue, floor of mouth, and alveolar margin (*Fig.* 32). There were enlarged cervical glands on the right side. Biopsy: squamous-celled carcinoma.

TREATMENT.—Ten needles at 10 mgrm. each of radium element (0·5 mm. of platinum) applied for 10 hours. Total dose 1000 mgrm. hours. This was followed by diminution in the size of the growth, but three months later, as the growth had not entirely disappeared, subperiosteal resection of the angle of the jaw was done previous to a second application of radium. This consisted of 10 needles at 5 mgrm. each (0·5 mm. of platinum) left in position for 33 hours. Total dose 1650 mgrm. hours. The condition of the tumour after treatment is shown in *Fig.* 33.

*Case* 4.—Mr. E. K., age 62. Nodular tumour the size of a shilling on the left lateral aspect of the tongue (*Fig.* 35), and ulceration of the left pillar of the fauces (*Fig.* 38). No palpable cervical glands. Biopsy of the tongue and pillar of the fauces shows both to be squamous-celled carcinomata. The lingual growth only was treated.

TREATMENT.—Eight needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum) inserted round the growth and left in position for 7 days. Total dose 667·2 mgrm. hours. Complete disappearance of both lingual and faucial neoplasms followed (*Figs.* 36, 37, 39). Six months later an enlarged gland was felt in the left submaxillary triangle. It was irradiated with 50 mgrm. of radium element (20 needles at 2 mgrm., 0·5 mm. of platinum; 2 needles of 5 mgrm., 0·5 mm. of platinum), in position for 10 hours daily for 5 days. Total dose 2500 mgrm. hours. No recurrence up to date.

*Case* 5.—Mrs. E. L., age 48. Small ulcer on left side of the tongue; no palpable cervical glands. Biopsy: squamous-celled carcinoma.

TREATMENT.—Six needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum screenage) applied for 5 days. Total dose 432 mgrm. hours. Entire disappearance of the growth followed. The patient remained well for ten months, then secondary deposits were found in the left submaxillary and anterior triangles. Excision of glands, which were found histologically to be epitheliomatous. Columbia paste collar (30 mm. thick) with 43·3 mgrm. of radium element in 23 foci (2 needles at 5 mgrm., 0·5 mm. of platinum; 8 needles at 2 mgrm., 0·65 mm. of platinum; 13 needles at 1·33 mgrm., 0·6 mm. of platinum) applied 18 hours daily for 21 days. Total dose 16367·4 mgrm. hours. Marked improvement of neck. Tongue normal.

*Case* 6.—Mr. F. B., age 65. Ulcer on tip and right border of the tongue (*Fig.* 40); no palpable cervical glands. Biopsy: squamous-celled carcinoma.

TREATMENT.—Ten needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum) applied for 5½ days. Total dose 792 mgrm. hours. Complete disappearance of growth followed (*Figs.* 41–43). Four months later there was a mass of malignant glands in the left submaxillary triangle; these were excised and found histologically to be epitheliomatous.

*Case* 7.—Mr. A. P., age 61. Papillomatous growth on under surface of the tongue and floor of the mouth (*Fig.* 44). Enlarged cervical glands palpable in the right anterior and submaxillary triangles. Biopsy: squamous-celled carcinoma.

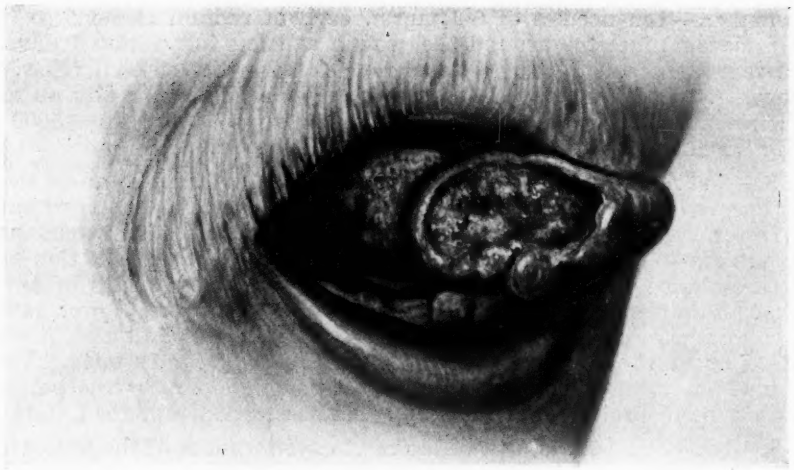

FIG. 40.—*Case* 6. Before treatment.

FIG. 41.—*Case* 6. Condition ten days after treatment.

FIG. 42.—*Case* 6. Condition one month after treatment.

FIG. 43.—*Case* 6. Condition four months after treatment.

TREATMENT.—Ten needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum screenage) inserted around the growth and left in position 7 days. Total dose 1008 mgrm. hours. Entire disappearance of growth followed (*Figs.* 45, 46). The neck was treated six weeks later by means of a collar carrying 40 mgrm. of radium element in 15 foci, 14 hours daily for 10 days. Total dose 5600 mgrm. hours. Entire disappearance of enlarged glands followed.

*Case* 8.—Mr. A. S., age 69. Extensive ulceration of the dorsum of the tongue extending deeply into the muscle and involving the whole of the anterior part of the organ. Wassermann reaction positive. Biopsy: squamous-celled carcinoma.

TREATMENT.—As no healthy tissue was available for the introduction of radium needles, these were applied to the surface of the growth by means of a vulcanite apparatus containing a lead lining protecting the upper and lower jaws. Ten needles at 0·6 mgrm. each of radium element (0·5 mm. of platinum), 5 needles at 1·33 mgrm. (0·6 mm. of platinum) applied 3 hours daily for 12 days. Total dose 215·40 mgrm. hours. Owing to the discomfort of the apparatus the patient could not tolerate a more prolonged application. The condition remained stationary.

*Case* 9.—Mr. F., age 63. Ulcer of the right anterior pillar of the fauces involving the tongue; no palpable cervical glands. Biopsy: squamous-celled carcinoma.

TREATMENT.—Six needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum) applied for 7 days. Total dose 600 mgrm. hours. Disappearance of primary growth followed. Secondary deposits in neck resected by Crile's method three months later. Death from cerebral thrombosis.

*Case* 10.—Mr. G. F. L., age 54. Extensive ulceration of right posterolateral aspect of the tongue involving the pillar of the fauces; large mass of secondary deposits in neck. Biopsy: squamous-celled carcinoma.

TREATMENT.—Crile's dissection of right side of the neck. Subperiosteal resection of ascending ramus of the lower jaw. Insertion of 10 needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum) for 5 days. Total dose 720 mgrm. hours. Death from bronchopneumonia on the ninth day. The tongue at the time of death showed marked diminution in size and changes in consistency of the growth.

*Case* 11.—Mr. J. C., age 51. Under the care of Mr. E. Rock Carling. Small ulcer on the under surface of the tongue and floor of the mouth; multiple enlarged cervical glands on both sides, but chiefly on the left side. Biopsy: squamous-celled carcinoma.

TREATMENT.—Wide dissection of cervical glands on the left side. (Histologically proved to be malignant.) Six needles of 1·33 mgrm. each of radium element (0·6 mm. of platinum) left in position for 5 days. Total dose 936 mgrm. hours. Complete disappearance of primary lesion followed. Columbia paste collar with 15·7 mgrm. of radium element in 11 foci applied for 5 days. Total dose 1884 mgrm. hours. The glands on the right side of the neck were dissected six months later and proved on histological examination to be inflammatory. The tongue was normal in appearance.

*Case* 12.—Mr. H. M., age 56. Under the care of Mr. Tudor Edwards. Ulcer on left lateral border of tongue posteriorly; no palpable glands in neck. Biopsy: squamous-celled carcinoma.

TREATMENT.—Eight needles of 0·6 mgrm. each of radium element and 1 needle of 1·33 mgrm. inserted around growth and left in position for 5 days. Total dose 735·6 mgrm. hours. Complete disappearance of growth followed. Dissection of cervical glands on left side fourteen days later. Histologically there was no evidence of malignancy in the glands.

*Case* 13.—Mr. G. C., age 74. Epithelioma of left side of the tongue. Secondary deposits in submaxillary glands and anterior triangle. Biopsy: squamous-celled carcinoma.

TREATMENT.—Eight needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum) inserted around the growth for 5 days. Total dose 576 mgrm. hours. Excision of submaxillary glands. Recurrence in carotid glands twelve months later. Columbia paste collar containing 40 mgrm. of radium element in 20 foci applied for 14 days, 10 hours daily. Total dose 5600 mgrm. hours. Neck normal.

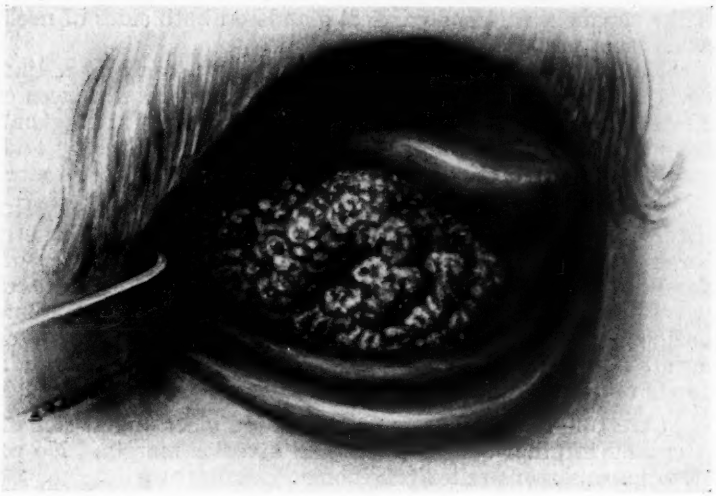

FIG. 44.—*Case* 7.
Before treatment.
Mobility of the tongue greatly diminished.

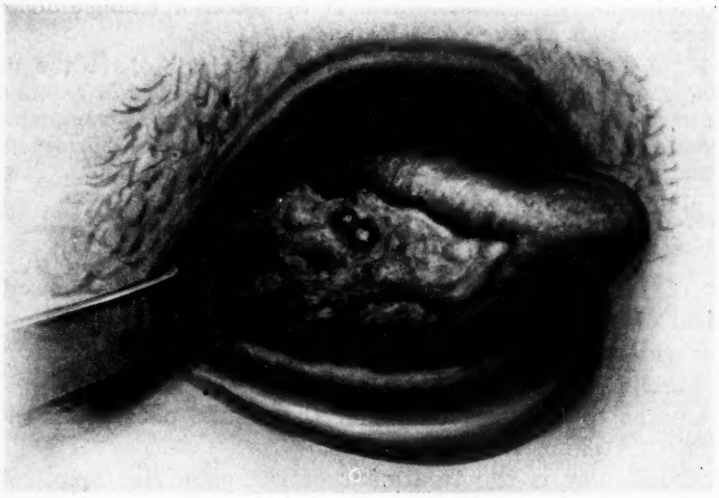

FIG. 45.—*Case* 7.
One month after treatment.
Mobility of the tongue returning to normal.

FIG. 46.—*Case* 7.
Condition three months after treatment.

*Case* 14.—Mr. McF., age 69. Ulcer of right pillar of fauces and adjacent portion of the tongue ; enlarged cervical glands on both sides of neck. Biopsy : squamous-celled carcinoma.

TREATMENT.—Six needles of 0·6 mgrm. each of radium element for 5 days. Total dose 432 mgrm. hours. Lesion of tongue and fauces completely disappeared. Bilateral Crile's dissection. Histologically malignant glands on right side only. Four months later secondary deposits in the right lung and mediastinum showing very plainly in a skiagram of the chest. No local recurrence in the mouth or neck.

*Case* 15.—Mr. F. S., age 54. Under the care of Mr. Rock Carling. Syphilitic history. Leucoplakia of the tongue for five years. Wassermann reaction positive. Large ulcer on right side of tongue, involving a quarter of the organ. Enlarged glands on right side of neck. Biopsy : squamous-celled carcinoma.

TREATMENT.—Ten needles of 1·33 mgrm. each of radium element (0·6 mm. of platinum) inserted around growth and left in position 4 days. Total dose 1248 mgrm. hours. Complete disappearance of growth followed. Excision of cervical glands refused by patient.

*Case* 16.—Mr. G. S., age 68. Fissured ulcer on left side of the tongue posteriorly extending to pillar of the fauces and alveolar margin. No palpable cervical glands. Biopsy : squamous-celled carcinoma.

TREATMENT.—Seven needles of 0·6 mgrm. each of radium element and 1 needle of 1·33 mgrm. inserted for 5 days. Total dose 660 mgrm. hours. Complete disappearance of growth followed. An abscess formed in the anterior triangle about three weeks later, and the pus was evacuated.

*Case* 17.—Mr. H. H., age 46. Very extensive ulcer on left side of the tongue from tip to pillar of the fauces, involving half the tongue. Wassermann reaction positive. No enlarged cervical glands palpable. Biopsy : squamous-celled carcinoma.

TREATMENT.—Eleven needles of 0·6 mgrm. each of radium element (0·5 mm. of platinum) inserted around growth for 7 days. Total dose 1108 mgrm. hours. The growth has entirely disappeared and the raw surface is gradually healing up.

## SUMMARY.

1. The treatment of 17* cases of cancer of the tongue is here reviewed. In each case the clinical diagnosis was confirmed by histological examination.

2. In 16 of these cases the primary growth in the tongue completely disappeared after treatment.

3. No claim is made as to the permanence of the results obtained ; the after-history of the patients alone can decide this.

4. The paramount difficulty is the treatment of the glandular area. A combination of surgical excision and radium therapy appears to be the most satisfactory method.

We cannot conclude this short account of some of the first cases treated with radium at Westminster Hospital without gratefully acknowledging the generosity of Mr. Austin Taylor, Chairman of the Hospital, and of the Directors of the Prudential Assurance Co., whose gifts of radium have made this work possible.

* Since this paper was written we have treated three other cases of epithelioma—one at the attachment of the lingual frænum to the floor of the mouth, one of the inside of the cheek, and one of the tongue—in each of which the primary growth has completely disappeared.

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# CARCINOMA OF THE ŒSOPHAGUS: TREATMENT BY DIATHERMY.

By A. J. WRIGHT,

SURGEON TO THE NOSE AND THROAT DEPARTMENT, BRISTOL GENERAL HOSPITAL.

Pathological Report by GEOFFREY HADFIELD,

PATHOLOGIST TO THE BRISTOL GENERAL HOSPITAL.

Until recent years but little had been done in cases of carcinoma of the œsophagus except the performance of a gastrostomy. The introduction of endoscopic methods of examination has gradually led to endoscopic methods of treatment. Carcinoma of the œsophagus is slow in growth, remains for a long time confined to the tube itself, is late to form glandular or metastatic deposits, and, owing to the obstruction produced, can sometimes be diagnosed fairly early. All these factors should make for success in removal, but difficulties of access have hitherto proved almost insurmountable. Until actual removal of the growth by open operation becomes the established method—if, indeed, it ever does—palliative methods must usually be employed. The simple performance of a gastrostomy leaves the individual in such a wretched condition that methods which aim at maintaining the patency of the œsophagus should, if possible, be perfected. Simple dilatation and intubation of the stricture, preferably with the metal tube introduced by Souttar, does do something to prolong life in a reasonable degree of comfort. The application of radium to the stricture is frequently successful in producing alleviation, but cures have not been obtained by most operators.

The very great utility of diathermy in the destruction of malignant growths in the mouth and pharynx—in which regions it has very largely supplanted the knife—has led me to try to adapt its use to malignant disease of the œsophagus. The problem for solution is very largely one of technique, and, after using the method here described for over two years, I feel justified in encouraging others to try it. While I cannot claim any cures, I am satisfied that, by the use of diathermy, one can usually prolong life and maintain a foodway, without any undue risk or suffering as a result of the operation. The use of diathermy, also, does not debar intubation; but if a tube is employed, it should not be inserted until the sloughs have separated—that is, for about ten days after operation. One advantage of diathermy over radium lies in the fact that the whole operation can be carried out in a few minutes. While cases are met with in which only one portion of the circumference of the tube is involved by the growth, in a very great majority this is not so. For the employment of diathermy in the œsophagus, a reasonable degree of proficiency in the use of the œsophagoscope is necessary.

## TECHNIQUE.

General anæsthesia with chloroform is employed, with a preliminary injection of hyoscine and atropine. Twenty minutes before the operation, the patient is given 20 min. of adrenalin diluted with ½ oz. of water to swallow.

This diminishes the tendency to hæmorrhage on touching the growth. It is helpful to use as large an œsophagoscope as possible. I employ two Mosher's oval tubes, of 2 cm. diameter, and 27 cm. and 45 cm. in length respectively. Suction, through a narrow tube attached to a suction apparatus and inserted down the œsophagoscope as required, is a great help, being much more rapid in action and producing less trauma than the use of mops. The special electrode employed (*Fig.* 47) is based on the bougies designed by Chevalier Jackson for use through the œsophagoscope. It consists essentially of an insulated shaft and handle, to the end of which various terminals can be screwed. For convenience I employ two shafts of 52 cm. and 70 cm. in length, the former for use with growths in the upper half, through the shorter œsophagoscope, and the latter for those in the lower half, through the longer tube. The terminals for screwing to the shaft consist of: (1) A disc 7 mm. in diameter; (2) A small spike; and (3) A gum-elastic bougie end, with, at its base, a conical metal ring which forms the actual terminal. A disc of heat-resisting composition is inserted between the metal terminal and the bougie end to prevent damage to the latter. This bougie electrode is made in three sizes, of 10, 15, and 20 French catheter gauge. A light cable facilitates manipulation.

FIG. 47.—Diathermy electrode for use in the œsophagus..

In the rare cases seen early, in which a nodule of growth involves one portion of the circumference, the disc or needle electrode can be applied to the growth under direct vision. In the very much more frequent cases, in which the whole or greater portion of the circumference of the tube is involved, producing a more or less annular stricture, the problem is a different one. I originally tried, in these cases, to destroy the growth from above downwards, but found that it was difficult, or impossible, to judge when one had reached the lower end of the growth, thereby introducing a risk either of not doing enough, or, on the other hand, of sloughing normal œsophagus below the growth. This led me to devise this special type of electrode. It is passed through the stricture under direct vision, and then drawn upwards until it meets with the resistance of the lower end of the growth (*Fig.* 48). The current is then switched on, and continuous traction is made on the instrument until it cuts its way out and comes into view at the upper end. The largest size of electrode which will pass with reasonable ease through the stricture should be employed. At least a fortnight should be

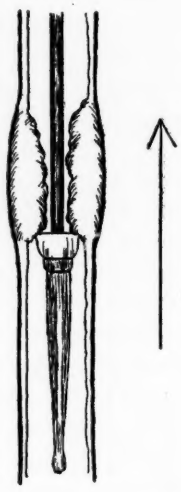

FIG. 48.—Electrode passed below the growth preparatory to treatment.

allowed to pass before deciding as to the result of any one application, and I think that if further applications are made, an interval of one month should be allowed to elapse between them.

In the selection of cases, each must be judged on its merits. Advanced cases, with an extreme degree of wasting, should have a gastrostomy performed, and, should their condition improve sufficiently, diathermy can be used at a later date. It should be remembered, in deciding the fitness of the patient for treatment with diathermy, that the whole operation can usually be carried out in a very few minutes and that the amount of shock produced is extremely slight. My own practice now is to employ diathermy in all cases in which the patient's condition is not desperate.

FIG. 49.—Extensive fungating carcinoma of the œsophagus sixteen days after the applications of diathermy. The arrows denote the site of the section illustrated in *Fig.* 50.

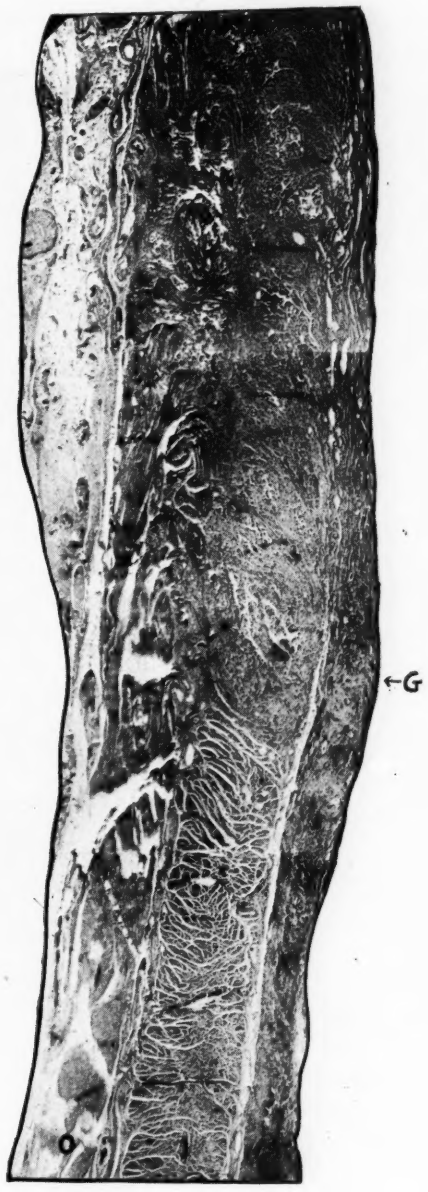


FIG. 50.—Low-power view of a longitudinal section of the œsophageal wall through the floor and edge of the ulcer which followed separation of the slough after diathermy had been applied to a projecting malignant mass. G, Islands of necrotic growth; 1, Inner muscular tunic; O, Outer muscular tunic.

## PATHOLOGICAL REPORT.

The specimen here described and illustrated (*Fig.* 49) shows the condition present seventeen days after a single application of diathermy to an extensive fungating growth in a woman, age 71. Death occurred as a result of sudden

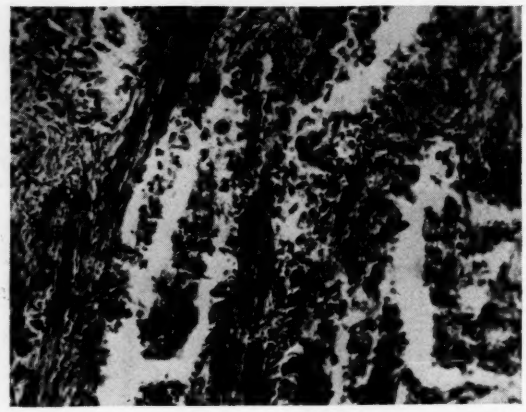

FIG. 51.—Inner muscular tunic below ulcer. Inflammatory reaction to diathermy is here relatively slight.

heart failure, and was not, apparently, in any way due to the diathermy. The pathological report is as follows :—

An irregular area of ulceration was found in the œsophageal wall at the level of the tracheal bifurcation. It was about 3 in. in length and involved $\frac{5}{6}$ of the circumference of the tube. The general level of the surface of the ulcer was that of the mucous membrane above and below it ; there was no

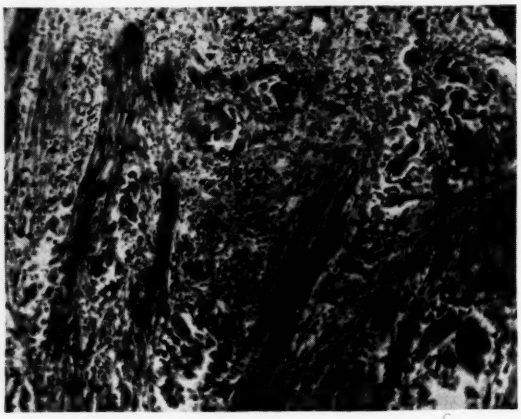

FIG. 52.—Inner layer of muscle below ulcer, showing masses of growth lying in the midst of inflammatory cells, the result of the diathermy.

stenosis. The surface of the ulcer was clean, and, except for three or four low nodules of growth, was red, soft, and spongy, and appeared to be composed of vascular granulation tissue of recent formation. The appearances on

section are illustrated in *Figs.* 50–53. *Fig.* 50 is a low-magnification view of about $1\frac{1}{4}$ in. of the œsophageal wall. In its lower third the muscle is intact. In the middle third the inner tunic is indistinct, acutely inflamed, and diffusely infiltrated by growth; *Fig.* 52 is from this part of the section. In the upper third both muscle coats are infiltrated and inflamed. The whole of the epithelial and submucous layer is replaced by a thick layer of inflammatory tissue, the result of the application of diathermy, the sloughs having all separated. Over the infiltrated muscle—that is, in the upper two-thirds of the section—the mucous and submucous layers contain many small islands of necrotic growth. Some of these are seen at G in *Fig.* 50, whilst *Fig.* 53 is a higher-power view of this layer at G. The scanty growth is seen to be embedded in inflammatory cells. Neither this nor any section showed any growth projecting above the general level of the mucous layer. The diathermy

FIG. 53.—Surface of ulcer. Small islands of necrotic growth lying in the midst of inflammatory cells.

has thus destroyed the mass of growth seen during life projecting into the lumen of the tube. Its effect on the muscle underlying the growth has been to cause a widespread inflammatory reaction, but whilst in some places the growth infiltrating the muscle is little affected (*Fig.* 51), in others it is almost 'snowed under' by inflammatory cells (*Fig.* 52).

## SUMMARY.

By the technique here described it is possible to employ diathermy in malignant disease of the œsophagus without undue risk, and with, at any rate, alleviation of symptoms.

# TREATMENT OF CARCINOMA OF THE ŒSOPHAGUS: BASED ON 100 PERSONAL CASES AND 18 POST-MORTEM REPORTS.

By H. S. SOUTTAR,
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The œsophagus is one of the common sites for the development of cancer, about 4 per cent of the cases of malignant disease being situated in this region. In the decade 1911-20 there were reported in England and Wales 15,909 deaths from carcinoma of the œsophagus, of which 12,059 were males. The annual mortality from the disease in this country is thus roughly 1600, of whom 1200 are males and 400 are females. It must then be admitted that the problem of its treatment is one of considerable importance, and this importance is increased by the lingering and miserable death from starvation to which, apart from surgical relief, these unfortunate people are condemned. The fact that the disease is invariably fatal is to some extent discounted by the advanced age at which it appears. But starvation is just as miserable an end at 70 as at 20.

Upon the pathological course of the disease and upon the best line of treatment the most varied opinions have been expressed. Some, impressed by the general absence of metastatic deposits, declare it to be of low malignancy and essentially suitable for attempts at radical removal, and one enthusiast has recently suggested that this might prove to be possible in 50 per cent of the cases. Others regard gastrostomy as a panacea, whilst others again, amongst whom may usually be numbered the patient himself and his friends, regard it as an invention of the devil. Under these circumstances it seemed to me worth while to undertake a fresh investigation of the problem from the strictly practical point of view of what could be done for the patient, and in the last few years my experiences have led me to certain very definite conclusions and to the adoption of an entirely new line of treatment. To state these conclusions without the evidence on which they are based would serve no useful purpose; but I shall discuss this evidence as briefly as possible, and then pass on to a more detailed description of the treatment I now adopt, together with the results so far obtained.

## ETIOLOGY.

In its incidence carcinoma of the œsophagus presents important peculiarities as regards sex, age, and site. (*See Tables I–III* and *Figs.* 54, 55.) It occurs four times as frequently in men as in women, but in the two sexes there is a remarkable contrast in the age and the site of its occurrence. In men it is a disease of later life, 96 per cent of the cases occurring after 45, and 88 per cent after 50, whilst the maximum incidence lies between 65 and 70. In women the incidence between 40 and 75 is about constant, whilst no

fewer than 8 per cent of the cases occur before 40. The difference is at least partly accounted for by the greater frequency of post-cricoid carcinoma in women, in whom it would seem to be related to a nervous spasm of the pharynx to which they are more liable than are men.

*Table I.*—Deaths from Carcinoma of Œsophagus for Ten Years 1911–20: England and Wales.

| | Under 40 | 40 45 | 45 50 | 50 55 | 55 60 | 60 65 | 65 70 | 70 75 | 75 80 | Over 80 | Totals | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Male .. | 124 | 375 | 972 | 1680 | 2146 | 2278 | 2058 | 1355 | 723 | 348 | 12,059 | 15,909 |
| Female .. | 289 | 315 | 415 | 464 | 470 | 507 | 464 | 414 | 282 | 230 | 3,850 | |

*Table II.*—Age Distribution in Own Series of Cases.

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Male .. | 0 | 3 | 6 | 11 | 14 | 17 | 15 | 10 | 3 | 1 | 80 | 100 |
| Female .. | 0 | 3 | 6 | 0 | 3 | 3 | 2 | 1 | 1 | 1 | 20 | |

*Table III.*—Site of Growth (Own Series of Cases).

| Inches from Teeth | Males | | Females | | Males | Females |
|---|---|---|---|---|---|---|
| | | | | | Per cent | Per cent |
| Upper 7 | 3 | | 3 | | | |
| Upper 8 | 5 | 10 | 3 | 6 | 13 | 31 |
| Upper 9 | 2 | | 0 | | | |
| Middle 10 | 28 | | 7 | | | |
| Middle 11 | 6 | | 2 | | | |
| Middle 12 | 10 | 59 | 0 | 10 | 74 | 53 |
| Middle 13 | 6 | | 0 | | | |
| Middle 14 | 9 | | 1 | | | |
| Lower 15 | 3 | | 0 | | | |
| Lower 16 | 5 | | 3 | | | |
| Lower 17 | 1 | 10 | 0 | 3 | 13 | 16 |
| Lower 18 | 1 | | 0 | | | |
| Total cases | 79 | | 19 | | + 2 unknown = 100 | |

To this circumstance is also due the greater incidence in women of carcinoma at the upper end of the œsophagus, where it occurs in 60 per cent of cases. In men it more commonly occurs at a lower level, and from large series of cases it would seem to occur at the upper, middle, and lower parts in the ratio of 1 : 2 : 3. In my own series of cases the incidence as regards site would seem to be somewhat different, 75 per cent of the male cases lying at a level of from 10 to 14 in. from the teeth (*Table III*). It must, however, be remembered that these figures refer to observations with the œsophagoscope, when only the upper surface of the stricture is seen. As many of the growths were several inches in length, the discrepancy is more apparent than

real. We thus see that carcinoma of the œsophagus is essentially a disease of elderly men, and that more than 80 per cent of the cases occur at or below the bifurcation of the trachea. In my own series in the great majority

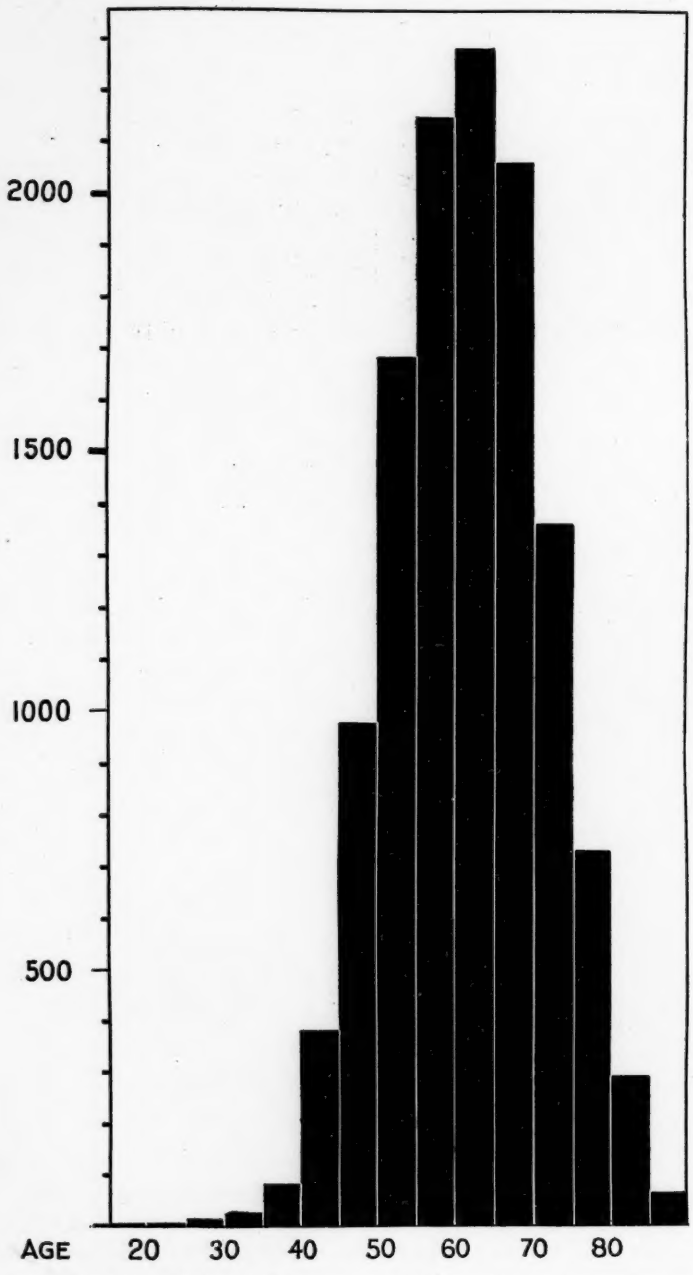


FIG. 54.—Deaths from carcinoma of the œsophagus for ten years—males.

of cases the upper limit of the growth lay approximately at this level—namely, 10 in. from the teeth. In women the disease is much less common,

it occurs more or less uniformly at any age over 40, and the level of its occurrence is more uniformly distributed.

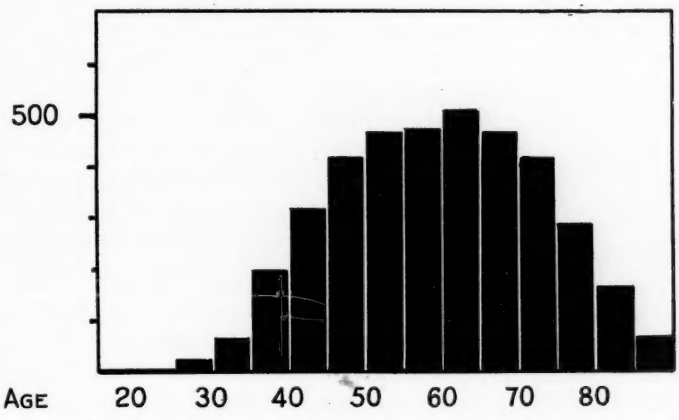


FIG. 55.—Deaths from carcinoma of the œsophagus for ten years—females.

## PATHOLOGY.

The most usual histological type of œsophageal carcinoma is squamous epithelioma, remarkable for the number of cell-nests and the degree of keratinization present. A rarer medullary form arises from the glandular structures of the œsophagus, and consists of rounded polygonal cells, often alveolar in arrangement, and of course with no cell-nests. Occasionally a columnar, type is seen, usually invading the œsophagus from the stomach but sometimes arising in other situations.

All these varieties of carcinoma spread by the lymphatics and tend to grow round the œsophagus and to produce stenosis. They may, however, remain for a long period localized to one portion of the wall, forming usually a deeply excavated ulcer with raised indurated margins, or a soft, vascular, fungating mass of growth: this is sometimes of great extent, involving in some cases as much as 6 in. of the tube, and even extending down to the greater curvature of the stomach. Though usually single, multiple nodules are sometimes seen, and these are of importance in that they may produce multiple strictures. They indicate extensive lymphatic permeation, since it is from the deep lymphatics and not by superficial spread that they have reached the mucosa.

No one can have examined these tumours post mortem without being struck by their great extent and by the havoc they have worked upon surrounding structures. Several inches of the œsophageal wall may be entirely destroyed, and we have had cases where for a considerable period the patient has swallowed through an intubated mass of carcinoma. Perforation of surrounding structures is naturally common, and the bronchi, pleura, mediastinum, or aorta may be opened up. The commonest termination is perforation of the œsophagus into the respiratory system, with the development of acute bronchopneumonia or of gangrene of the lung.

Secondary deposits occur in the lymph glands along the œsophagus, and

they may extend upwards to the neck or downwards to the epigastrium, giving rise sometimes to large palpable masses. In the neck extensive infiltration may occur and may produce stenosis at this site before the primary growth at a lower level gives any evidence of its presence. Secondary deposits at distant sites are unusual, and of this the local pathology of the growth is a sufficient explanation. Growing in a highly vascular region and surrounded by vital structures, the primary growth destroys the individual before there is any opportunity for the development of secondary deposits.

This is scarcely a picture of a growth of low malignancy lending itself to easy removal, but its accuracy may be judged from the records of post-mortem examinations given below. The objection will be raised that this is a picture of the terminal stage, and that at an early stage it would be very different. The objection is quite valid, but entirely illusory, for the picture drawn accurately represents, in many instances, the stage at which symptoms first appear. Indeed, as will be seen from the records below, carcinoma of the œsophagus may exist in an advanced stage without a single local symptom to indicate its presence. In addition, the appearance seen with the œsophagoscope at the most a few weeks after the onset of symptoms, entirely corroborates the picture I have drawn. No one can have made extensive use of this instrument without often being astounded at the extent of the lesions which are compatible with life, and sometimes even with apparent health. My own experience points to the definite conclusion that carcinoma of the œsophagus is a disease of high malignancy, and that symptoms appear so late that radical removal, except in unique cases, is a pathological impossibility.

The pathological anatomy of carcinoma of the œsophagus cannot be better illustrated than by a series of post-mortem examinations, and for this purpose I have taken 18 consecutive autopsies from the records of the London Hospital between the years 1916 and 1922, for which I would express my grateful thanks to Professor Turnbull. It might be imagined that these were all cases with symptoms of long duration, and indeed it is quite evident from the recorded findings that the disease itself had been present for a long time. As regards symptoms, however, it is a very remarkable fact that the average duration was only 4·7 months, whilst the average duration of symptoms in my own series of cases *at the time of the patient's first appearance* was 4·8 months. It would seem then that I was dealing on the average with cases at a later stage than those which came to post-mortem examination. This was, of course, a statistical accident, but it strongly supports my contention that these post-mortem reports may fairly be taken as the pathological picture, only very slightly overdrawn, of the clinical material with which I had to deal, and it is in this that their great value lies. Terrible though they are, these reports present to us remorselessly accurate pictures to which the true pathology of the living can only be the penultimate stage.

The causes of death in these cases were as follows :—

| | | | |
|---|---|---|---|
| Invasion of lung .. .. .. .. | 4 | Subphrenic abscess and bronchopneumonia .. .. .. .. .. | 1 |
| Perforation of bronchus .. .. | 3 | Secondary in brain .. .. .. | 1 |
| Perforation of trachea .. .. .. | 3 | Exhaustion and bronchopneumonia .. | 4 |
| Perforation of aorta .. .. .. | 1 | | |
| Hæmorrhage from growth .. .. | 1 | | |

Thus 10 of the 18 cases died from direct involvement of the respiratory tract, and 5 more from its involvement indirectly, making a total of 15, or 83 per cent, who died from pulmonary complications. Of the remainder, 2 died from hæmorrhage and 1 from a secondary deposit in the brain. The last is a rare complication, so that we shall not be far out if we say that out of every 10 cases 9 will die from purulent bronchopneumonia and 1 from hæmorrhage.

**POST-MORTEM REPORTS OF 18 CASES.**

*Case* 1.—Edward M., age 55. Six months' increasing dysphagia, regurgitation of solids, increasing pain, wasting. Refused gastrostomy.

Post Mortem.—A large ulcerated polypoid growth 12 cm. long. It extended from the cardiac orifice upwards along the œsophagus and downwards into the wall of the stomach. A perforation opened into a large abscess cavity below the diaphragm. No secondaries were discovered. Death from bronchopneumonia. Sections showed œsophageal carcinoma of the alveolar medullary type.

*Case* 2.—Francis C., age 47. Four months' increasing dysphagia. Refused gastrostomy. Died suddenly.

Post Mortem.—A fungating carcinomatous ulcer, 10 cm. long by 8 cm. in circumference, completely encircled the œsophagus below the bifurcation of the trachea. A large perforation in the commencement of the descending thoracic aorta communicated with the œsophagus. Secondary deposits were found in the adjacent glands, but nowhere else. The stomach and intestines contained very large quantities of blood. Death from hæmorrhage.

*Case* 3.—Georgina S., age 63. Three months' pain in epigastrium through to back, not related to meals. Stomach tube for test meal passed without difficulty. Died with pulmonary symptoms.

Post Mortem.—A fungating carcinomatous ulcer, 11 cm. long by 8 cm. in circumference, had almost completely encircled the œsophageal wall below the bifurcation of the trachea. It had invaded the aorta and the mesial aspect of the lower lobe of the right lung. Death from bronchopneumonia.

*Case* 4.—John C., age 72. For some months increasing dysphagia and wasting. Died two days after gastrostomy.

Post Mortem.—An ulcerated carcinoma, 5 cm. diameter, with fungating edges, almost entirely encircled the œsophagus at the cardiac opening and involved the stomach. There were secondary growths in the adjacent glands and a small nodule on the lower lobe of the left kidney. Death from bronchopneumonia.

*Case* 5.—Arthur E., age 46. Ten weeks' increasing dysphagia and three days' absolute obstruction. He died six weeks after a gastrostomy, which had completely closed.

Post Mortem.—An ulcerated carcinoma, 4·5 cm. long, completely encircled the œsophagus immediately above the bifurcation of the trachea. It involved the upper lobe of the left lung, and a perforation communicated with a large cavity in the left lobe containing food remains. There were no secondary deposits. Sections showed scirrhous epithelioma.

*Case* 6.—Arthur T., age 61. Four months' bronchitis and dysphagia. On admission very ill with paroxysmal dyspnœa.

Post Mortem.—A huge carcinomatous ulcer, 13 cm. long, occupied the whole of the œsophagus below the bifurcation of the trachea and extended on to the stomach. It had produced no constriction of the œsophagus, and the growth was nowhere more than 0·7 cm. thick. A large portion of the floor was formed by the pericardium, into which a flat nodule of growth projected. The hilum of the left lung was infiltrated, and there was an abscess at this point. There were no secondary deposits.

*Case* 7.—Henry T., age 44. Seven weeks before admission he received a heavy blow on the left side of the chest, and soon after vomited up blood. He had since

suffered from increasing dysphagia, and was now unable to swallow. He died two days after gastrostomy.

POST MORTEM.—An ulcerated carcinoma with fungating edge, 8 cm. by 4 cm., encircled the œsophagus below the bifurcation of the trachea. It had involved the left bronchus, into which there was a large perforation. Death from bronchopneumonia.

*Case* 8.—Edward W., age 45. Six months' increasing dysphagia. Died one month after gastrostomy.

POST MORTEM.—A fungating carcinomatous ulcer, 12 cm. by 8 cm., extended downwards from the tracheal bifurcation, almost completely encircling the œsophagus. A perforation at the middle of the growth led into a cavity in the lower lobe of the right lung, and the pericardium was involved by direct extension. Secondary nodules had invaded the 7th cervical and the 1st dorsal vertebræ, and the œsophagus itself at a higher level. There was a secondary deposit in the left suprarenal. Sections showed a squamous horny carcinoma.

*Case* 9.—Ernest B., age 57. For three months, cough, rapid wasting, and regurgitation of food. For six weeks swallowed liquids only.

POST MORTEM.—A carcinomatous ulcer, 5 cm. by 1·5 cm., almost encircled the œsophagus below the bifurcation of the trachea. There was a perforation into the left bronchus. Death from bronchopneumonia.

*Case* 10.—Alfred K., age 56. Three months' loss of appetite, regurgitation of food, and wasting. Died two days after gastrostomy.

POST MORTEM.—An ulcerated carcinoma, 10 cm. long, encircled the œsophagus below the bifurcation of the trachea, extending to the cardiac orifice and producing marked constriction. There were secondary deposits in the glands.

*Case* 11.—William S., age 49. Seven months' increasing dysphagia, which became complete. He died one month after gastrostomy.

POST MORTEM.—A large carcinomatous ulcer, 6 cm. diameter, encircled the œsophagus just above the cardia. There were secondary deposits in the glands.

*Case* 12.—Eugenie T., age 48. Eight months' soreness of right side of neck. Four months' dysphagia and cough, with severe dyspnœa and rapid loss of weight.

POST MORTEM.—A carcinomatous ulcer commencing in the right pyriform sinus involved the upper end of the œsophagus. Death from purulent bronchopneumonia. Sections showed squamous carcinoma with large cell-nests.

*Case* 13.—John D., age 67. Ten weeks' increasing dysphagia, pain in chest, and gross loss of weight.

POST MORTEM.—An ulcerated carcinoma, 7 cm. long, above bifurcation of trachea, with tight constriction. A nodule of carcinoma projected into the lumen of the trachea. Sections showed squamous carcinoma.

*Case* 14.—Minnie B., age 45. Three months' increasing dysphagia. Death from hæmorrhage.

POST MORTEM.—An ulcerated carcinoma, 5 cm. long, completely encircled the upper œsophagus, extending into the pharynx. Hæmorrhage from œsophagus into stomach. Squamous carcinoma.

*Case* 15.—John G., age 62. Six months' history of tumour in left centrum ovale. No œsophageal signs. Death in coma.

POST MORTEM.—Carcinomatous ulcer, 7 cm. by 3 cm., in œsophageal wall below tracheal bifurcation. Secondaries in the glands, and large secondary nodules in the centrum ovale, superior vermis, and the left lobe of the cerebellum. Sections showed a polygonal carcinoma with no tendency to the formation of nests. The secondaries in the brain were very vascular, and the destruction of the brain tissue was striking.

*Case* 16.—John E., age 54. Seven months' increasing dysphagia and rapid wasting. For two months taking food produced violent coughing. Died ten days later after gastrostomy.

POST MORTEM.—Carcinomatous ulcer, 7 cm. long, encircling œsophagus at

upper end. It invaded the posterior wall of the trachea, which was perforated. There were secondary nodules in the glands. Squamous carcinoma with cell-nests.

*Case* 17.—Thomas M., age 50. Three months' regurgitation of food and wasting. Death with pulmonary symptoms.

Post Mortem.—Carcinoma, 11 cm. long, completely encircling the œsophagus below the bifurcation of the trachea. The left bronchus was perforated. No secondaries.

*Case* 18.—Alfred S., age 48. Nine months' increasing dysphagia. For three months, regular dilatation by bougies. Died two days after a sudden attack of dyspnœa with pulmonary signs.

Post Mortem.—A ragged carcinomatous ulcer, 8 cm. by 3 cm., had destroyed the wall of the œsophagus opposite the bifurcation of the trachea, which was perforated. A large nodule of growth projected into the trachea itself. There were extensive secondaries in the glands. Squamous carcinoma with little nest formation.

## SYMPTOMS.

A steadily increasing dysphagia is undoubtedly the primary symptom of œsophageal carcinoma, and where this occurs in a man past middle age there can be little doubt as to the diagnosis. Occasionally the onset is quite abrupt, and the patient is under the impression that a piece of bone has stuck in his throat. His symptoms are probably the result of reflex spasm and may disappear for a time, to reappear as a persistent difficulty in swallowing. Naturally solids are first affected, but in most cases it will be noted quite early that even liquids are checked and only pass down slowly, whilst the regurgitation of unaltered food occurs from the earliest stage. The irritation of the growth produces a secretion of viscid mucus which fills the œsophagus and still further impedes swallowing. Often the patient is entirely unable to swallow in the morning until he has ejected a quantity of white froth, after which he may be able to take food without much difficulty. I have come to regard this white froth as a diagnostic feature. Sometimes it is blood-stained and foul, and it then indicates a fungating growth with extensive ulceration. Occasionally the horrible fœtor indicates gangrene of the lung.

Acute pain, apart from perforation, is a rare symptom, but a dull discomfort referred to the upper sternum is quite common. It is accentuated by swallowing, and it may be very persistent and wearing. Involvement of the dorsal roots may lead to severe intercostal neuralgia, and in one of my cases this appeared as quite an early symptom. Involvement of the recurrent laryngeal nerves may lead to hoarseness and aphonia, and it is important to remember that, if both nerves are involved, bilateral abductor paralysis may result in paroxysms of dyspnœa of the utmost gravity. It is said that persistent hiccup may be an early symptom in a low carcinoma from involvement of the phrenic nerves.

Many important symptoms arise from involvement of the respiratory passages. Irritation produces the secretion of a thick tenacious mucus which may be a source of much annoyance, and which, when swallowed, produces the froth to which we have referred. Invasion of the tracheal wall often occurs, and gives rise at first to respiratory embarrassment from the swelling of the mucosa which results. A nodule of growth may actually perforate the wall and obstruct the lumen, whilst ulceration may lead to a fistulous

communication between the trachea and the œsophagus. When this has occurred, swallowing may give rise to violent fits of coughing, more marked with liquids than with solids, and a septic bronchopneumonia inevitably follows.

Wasting is almost invariable, and is the direct result of starvation. Where ulceration and sepsis are present there may be definite cachexia and anorexia, but many of these patients are ravenously hungry and improve rapidly in condition as soon as they are enabled to swallow. They contrast in a striking manner with cases of carcinoma of the stomach, and it is only in the later stages of exhaustion that they present the shrunken cachectic appearance characteristic of that disease. They are, however, very miserable from their inability to swallow, and thirst, hunger, and sometimes a constant irritating cough rapidly sap their vitality.

## EXAMINATION AND TREATMENT.

In examination of these cases we rely on two methods, radiography and œsophagoscopy. By means of the former we can establish the site of the stricture and its organic nature, and in general we can tell whether it is due to malignant disease of the œsophagus or to some other condition. With

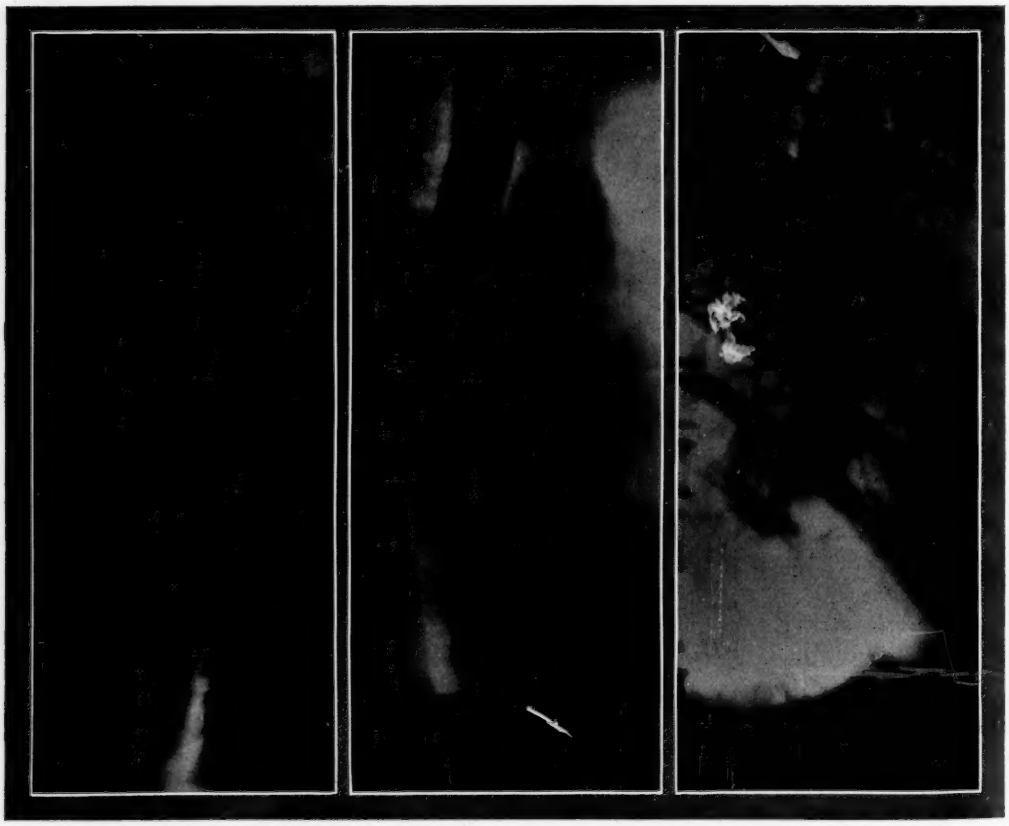

FIG. 56.—Carcinoma of the œsophagus. Typical radiographs.

the œsophagoscope we can examine its pathological appearance and investigate its permeability, and in the event of its proving to be permeable by a bougie we can institute treatment.

**X-ray Examination.**—This should be carried out in two parts, by screen examination and by the study of a film, since the latter gives much more detailed information. The patient stands with the right arm raised above his head, and he is placed somewhat obliquely so that the rays pass either from left dorsal to right ventral or vice versa. In this way the shadow of the heart and great vessels and of the spine are avoided, and a clear picture of the œsophagus itself is obtained. He now swallows a thick emulsion of barium sulphate of the consistency of viscous syrup, and the observer watches on the screen the passage of the emulsion through the œsophagus. It will be seen to reach the obstruction, to fill up the œsophagus above it, and then to trickle slowly through the stricture, which will almost always allow of its slow passage. The appearances with a malignant stricture are absolutely characteristic. There may be a very moderate degree of dilatation above, giving a solid shadow which terminates in a cone pointing downwards, and from the apex of this cone a fine twisted stream of barium can be seen threading the tortuous channel of the growth. Below this the shadow of the normal œsophagus is again observed as the emulsion adheres to its walls. So absolutely characteristic are the appearances that in nine cases out of ten the diagnosis can be established by this means alone. (*Fig.* 56.)

**Œsophagoscopy.**—This ought to be regarded as a procedure demanding the highest technical skill, for although the expert can avoid trouble, in inexpert hands there is no more dangerous proceeding. It must be remembered that the œsophagus is in any case far more fragile than the intestine,

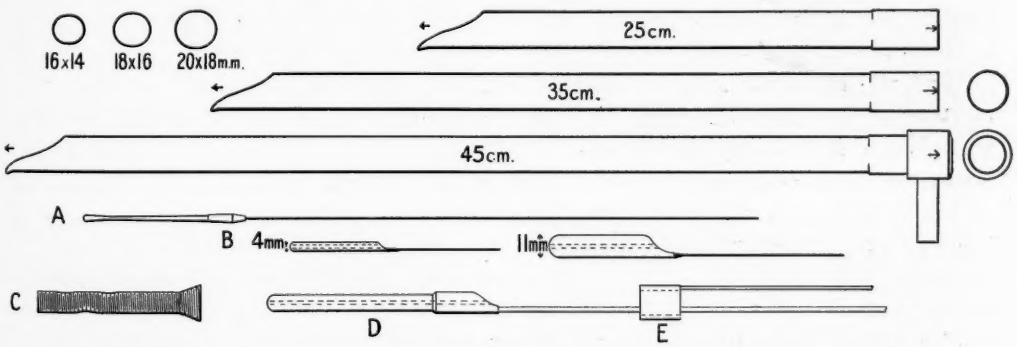


FIG. 57.—Special œsophagoscopes and accessory instruments for intubation. A, Flexible bougie; B, Aluminium dilators, to pass over shaft of A; C, Flexible wire tube; D, E, Introducer and releasing ring.

from the absence of a submucous coat; but we are here dealing with elderly men, and with a tube the walls of which may be deeply ulcerated or may have been replaced by a soft mass of fungating growth. Unless the utmost gentleness is exercised, fatal damage may be done, and even if this is avoided, bleeding may be caused which will so far obscure the view as to render the examination nugatory.

For various reasons I have not found the ordinary instruments suitable

for my purpose, and I have therefore had a special set constructed. It consists of oval tubes of various dimensions fitting interchangeably into the same Brünings handle with a proximal light. The tubes are in three sizes, 20 by 18, 18 by 6, and 16 by 14 mm., outside dimensions, and for each size three lengths, 10, 14, and 18 in., are provided. It is thus always possible to use the largest tube which can be passed on the patient and the shortest which will reach the stricture, so that all procedures are carried out under the best conditions obtainable. (*Fig.* 57.)

Either local or general anæsthesia may be employed. In either case the patient should be given an injection of morphia and atropine half an hour earlier, and just before the operation the epiglottis and pharynx should be painted repeatedly with 10 per cent cocaine. With these precautions the manipulations required can be carried out with very little inconvenience, and even where a general anæsthetic is given to allay the patient's anxiety, these preliminary measures are of great assistance in abolishing spasm.

With the patient lying flat on his back, the head is extended at the occipito-atlantal joint, but is lifted forward so as to straighten the cervical spine. No greater mistake can be made than forcibly to extend the neck. The head, supported by an assistant, is now slightly deflected to one side, and the operator inserts his instrument at the angle of the mouth, thus

FIG. 58.—A, *Case* 90. Growth completely encircling œsophagus. Tight stricture. Absolute obstruction. B, *Case* 90. Tube in place. Functional success immediate. C, *Case* 81. Soft fungating growth, with secondary nodule at higher level. D, *Case* 81. After intubation with tube and rubber cone. Note secondary nodule above level of tube. E, *Case* 87. Tight carcinomatous stricture just below cricoid. F, *Case* 83. Fungating growth on posterior wall at cardiac orifice. Guide has been passed into stomach.
(*Drawings by Mr. F. A. Sewell.*)

avoiding the prominent central incisors, if they are present, and entering at a point much nearer to the pharynx.

Under the control of vision the epiglottis is now lifted forwards on the beak of the instrument, which enters the pyriform fossa of the opposite side. From this it is deflected to the middle line, where it is probably brought abruptly to rest by spasmodic closure of the cricopharyngeus, the passage of which is the most difficult part of the operation. Gently pressing the tip of his instrument against the sphincter, the operator waits until with a deep inspiration it opens, disclosing the lumen of the cervical œsophagus, into which the instrument glides. After advancing an inch further the wider lumen of the thoracic œsophagus opens up, expanding with each inspiration and closing on expiration to a transverse slit. The assistant now slightly raises the head, and the instrument glides easily through the thoracic œsophagus, following the natural concavity of the thoracic spine. As the lip of the instrument, guided by the vertebral column, again turns forwards, the head, neck, and shoulders must be depressed. The lumen now assumes the appearance of a vertical slit where the œsophagus passes through the diaphragm. At this point extreme care must be taken, for not only is the œsophagus itself here very fragile, but its fixation to the diaphragm renders it peculiarly liable to trauma. Beyond this the cardia will be seen as a puckered opening to which folds of mucosa converge. Entry into the stomach

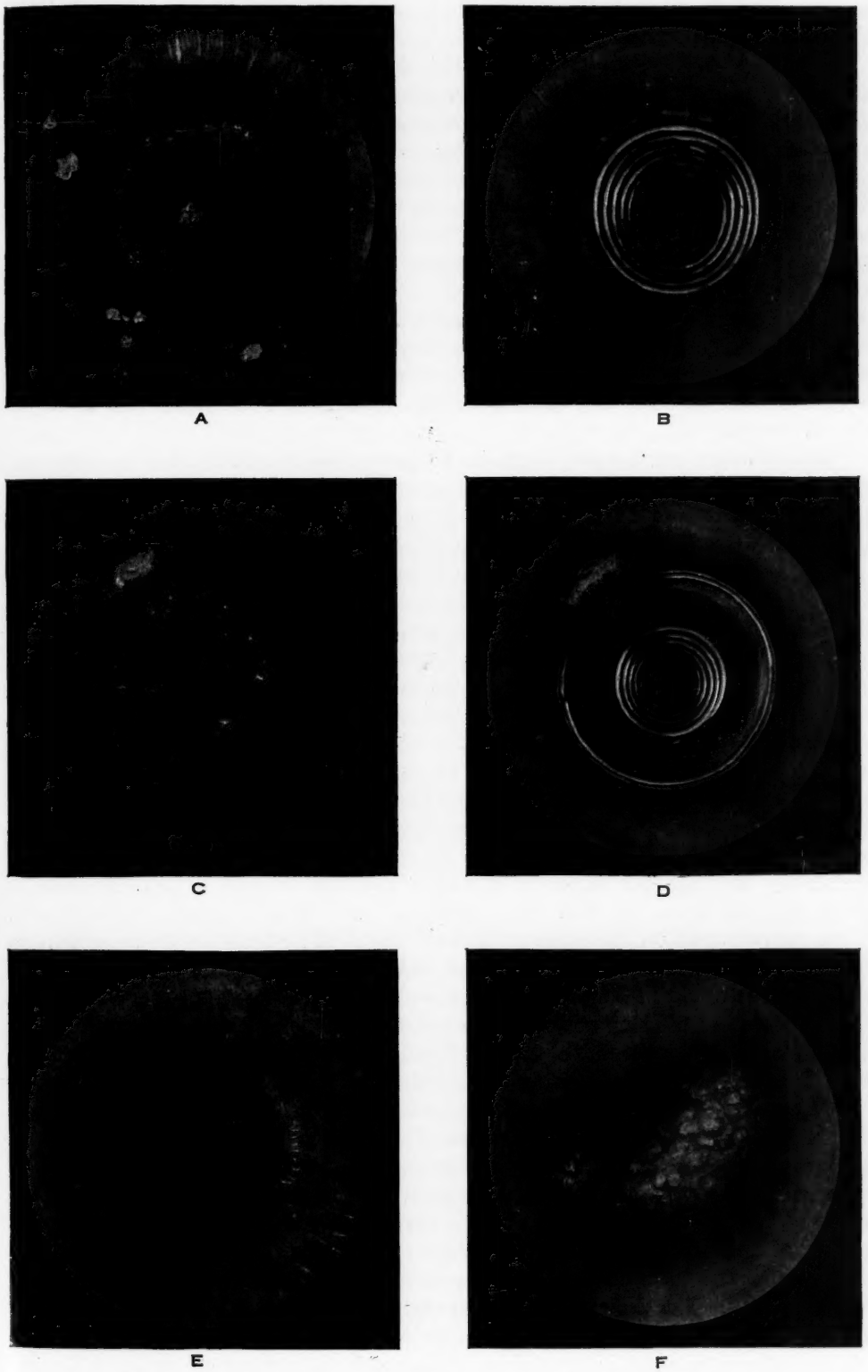


FIG 58. Œsophagoscopic appearances in carcinoma. For details *see* p. 86.

is announced by a gush of fluid and the appearance of heavy folds of dark-pink mucous membrane.

Where a carcinoma is present the appearances are very characteristic (*Fig.* 58). On entering the œsophagus, its lumen will be seen to be occupied by a thick white, frothy mucus which entirely blocks the view. On advancing farther, variable quantities of liquid and of food remains appear, and if these are excessive the simplest plan for their removal is to tip up the table and to allow them to flow down the instrument by gravity. As the stricture is approached, it will be seen that the walls of the œsophagus, thickened by infiltration, no longer open and close with respiration, whilst slight dilatation of the lumen is usual. The stricture itself may appear as a mere constriction of the mucosa or as a fungating mass of vascular growth which bleeds readily on contact, or we may see a deep ulcer in the œsophageal wall. In any event it must be remembered that all we see is the upper margin of the growth, and that from the appearances alone we can form no idea of its extent.

An attempt must now be made to pass a bougie through the stricture, and for this purpose I use a modification of Jackson's bougie, a flexible sound (*Fig.* 57, A) attached to the end of a steel wire which does not interfere with vision. Under the guidance of the eye the end of the bougie is manipulated until it engages in the lumen of the stricture, though in most cases it will then pass without difficulty. Not the least force must be used, for the wall at this point may be formed by soft growth through which, with the slightest violence, the bougie may penetrate. Once the stricture has been successfully passed, the bougie is pushed on until the wire occupies the stricture and forms a guide over which the dilators may be passed.

These dilators (*Fig.* 57, B) are of a novel type which I devised specially for this purpose. They are formed of short cylindrical rods of aluminium, with an axial hole to slide freely over the wire guide. Each is attached to a long rigid steel wire, and the sizes rise by half millimetres from 4 mm., the smallest, to 11 mm., the largest, in diameter. By passing these in turn completely through the stricture it can usually be dilated with great facility up to the full size. At the same time, as each dilator passes through and clears the constriction, an accurate measurement of its length may be obtained. The method appears to be devoid of risk, but there is one circumstance in which I have always felt it necessary to exercise great caution. If, on dilating a dense stricture offering considerable resistance, it is noted that the resistance has suddenly disappeared, dilatation should proceed no farther, as obviously there is a distinct risk of tearing the œsophageal wall if it is continued.

Mere dilatation will often give great relief, but in most cases the dysphagia soon recurs. To overcome this I devised a tube formed of a coil of German-silver wire, which has proved most satisfactory. A cone-shaped upper end rests on the top of the stricture, and to prevent upward movement the tube is made oval in section and a spiral twist is given to the oval. The success of this last device has been very remarkable, for not a single tube in all my series has been displaced upwards. In soft growths, on the other hand, the whole tube has frequently passed on, dilating the œsophagus to such a degree, however, that in most cases swallowing continued. The tube passes

through the bowel without incident. From its extreme flexibility the tube is readily tolerated, the patient being quite unconscious of its presence, whilst its structure allows of a lumen which can be obtained in no other way. I have never seen any evidence of ulceration of the wall of the œsophagus by the pressure of the tube, and indeed, if properly inserted, its contact with the mucosa is so slight that this is not likely to occur. The tube, in fact, generally tunnels a mass of carcinoma, and in this case ulceration ceases to have its normal meaning. In one of my patients, a man of 69 with a carcinomatous stricture 12 in. from the teeth, a tube remained in place, repeatedly verified by X rays, for eleven months, the patient swallowing well and being entirely ignorant of the existence of the tube.

The method of insertion is as follows. After dilating the stricture as far as is considered safe, a tube of suitable size (*Fig.* 57, C) is selected and is threaded on to an introducer (*Fig.* 57, D). This slides over the guide wire (*Fig.* 57, A), which is still in place, and serves to steer the lower end of the tube through the stricture. The introducer and the guide bougie are now withdrawn through the ring (*Fig.* 57, E), which is then itself withdrawn, and the tube is left in place, its expanded upper end resting on the face of the stricture. The method is applicable to strictures in all situations except the cervical œsophagus, where the irritation of the trachea by the tube prevents its toleration. On several occasions I have passed a tube through a stricture of the cardiac opening into the stomach with a satisfactory result. On the other hand, where a growth is limited to one portion of the œsophageal wall it is useless to insert a tube, as it will be passed at once; these cases, however, are easily relieved by dilatation. (*Figs.* 59, 60, 61.)

Where the tumour is very soft and friable, difficulty may be experienced from the early passage of the tube into the stomach. To avoid this I use a small cone of rubber. This is passed through the œsophagoscope and rests on the top of the growth, and through the cone the wire tube is now passed. The manipulation is by no means easy, but the result is extremely satisfactory, and the method has enabled me to deal with a most difficult class of case. (*Fig.* 62.)

## RESULTS.

In the large majority of cases the immediate result of intubation is extremely satisfactory. With few exceptions the patients are able next day to swallow liquids and soft foods with perfect facility. In the case of an emaciated man who has been starved for weeks it is necessary to proceed with caution, but the average patient leaves the hospital two days later having had two poached eggs, fried bread, toast, and coffee for breakfast. As several of my patients had for some days been unable to swallow even the smallest drop of fluid, the change is a very remarkable one, and in most cases the relief lasts for the lifetime of the patient.

In a series of 98 cases I have performed intubation on 50 occasions, and the statistical results are as follows: 7 patients died soon after the operation, but in only two of these was there any reason to suppose that death was due to the manipulations involved. In one of these, a case with extreme emaciation, it was thought that the œsophagus was torn in inserting

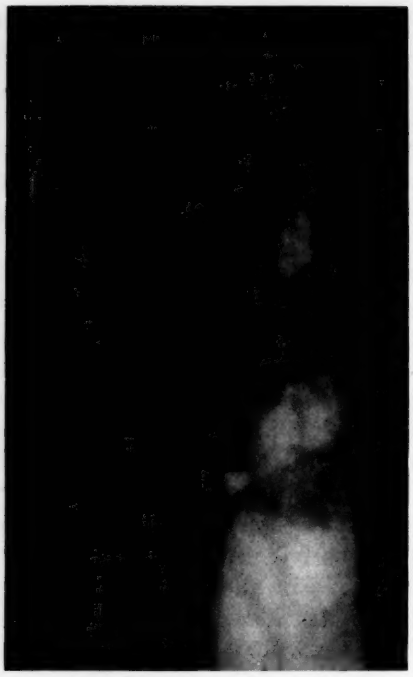

FIG. 59.—Tube in carcinomatous stricture.

FIG. 60.—Intubated carcinomatous stricture. Passage of barium meal.

FIG. 61.—Carcinoma of cardiac end. Long stricture dilated by tube which actually enters the stomach.

FIG. 62.—Tube in position with rubber flange.

the tube; in the other the end of the tube had perforated the base of a large carcinomatous ulcer. In the remaining 5 cases, one death was due to exhaustion, one to mediastinal suppuration, one to subphrenic abscess, one to perforation of the lung, and one to perforation and gangrene of the lung. In each case it was ascertained that the cause of death was not the result of the intubation, but occurred in the natural course of the disease. The true operation mortality of this series is therefore 4 per cent, and one may hope to avoid even this by greater experience.

As to the duration of life after intubation or dilatation followed by intubation, in 26 cases followed to a conclusion this varied between 2 weeks and 23 months, the average being 5·3 months. Of the other cases, some have been lost sight of, but many are still alive, in good health after periods up to 7 months. In those who have died, death has resulted from pure exhaustion or from one of the complications to which we have already referred. In some dysphagia recurs, and in these we have on several occasions demonstrated an extension of the growth above the level of the tube. But most of the patients had swallowed satisfactorily till near the end.

As compared with these figures, gastrostomy was performed in 20 cases with 7 deaths, in each case with some complication unconnected with operative technique. The average duration of life of the survivors in 10 cases, followed up was 3·6 months.

Without unduly stressing these figures, I think they prove that intubation is far safer than gastrostomy, and that it affords a longer expectation of life. From the patient's point of view, however, two methods could scarcely be in greater contrast. After gastrostomy he is an invalid, kept alive by artificial feeding and debarred from the exercise of a function without which life is not worth living. After a successful intubation by this method he can eat and drink almost like a normal man, he can enjoy a meal with his family, and there is nothing objective to remind either himself or his family of his disease. I think it reasonable to hope that with further experience such a result will in most cases be obtainable, and it seems to be all that one can ask in a disease which I am firmly convinced is entirely beyond the possibility of surgical cure.

## CONCLUSIONS.

This study, based on 100 clinical cases and 18 post-mortem examinations, appears to me to prove quite clearly that carcinoma of the œsophagus is a highly malignant form of growth; that it only fails to produce widespread secondary deposits because it destroys the individual at such an early date; that its earliest symptoms, usually those of obstruction, only appear at a late stage of the disease; and that it therefore neither is nor ever can be amenable to direct surgical attack, except in cases so rare as to be of no practical importance.

It would seem that all we can hope to do is to give the patient relief from dysphagia, and that this is best accomplished by intubation. The method of intubation here described has the advantage that in most cases the patient is able to swallow ordinary food, and in a large number of cases he retains this power until death, which occurs usually from direct involvement of the respiratory tract.

*Table IV.*—Treatment and Results in 100 Cases of Œsophageal Carcinoma.

| Number | Sex and Age | Duration (Months) | Site (Inches from Teeth) | Treatment | Survival (Months) | Remarks |
|---|---|---|---|---|---|---|
| 1 | F. 50 | 2·5 | 16 | Nil | 3 | Refused gastrostomy |
| 2 | M. 63 | 3 | 14 | Gastrostomy | Died | Bronchopneumonia |
| 3 | F. 43 | 9 | 7 | ditto | 8 | |
| 4 | M. 60 | 3 | 11 | ditto | Died | Hæmorrhage from œsophagus |
| 5 | M. 46 | 3 | 7 | Gastrostomy, tracheotomy | 6 | Emergency tracheotomy. Large mass involving pharynx, larynx, and œsophagus |
| 6 | M. 59 | 3 | 13 | Dilatation | ? | Swallowed fluids easily on discharge |
| 7 | M. 64 | 3 | 12 | Nil | ? | Refused gastrostomy |
| 8 | F. 48 | 8 | 8 | Gastrostomy | Died | Gangrene of lung |
| 9 | M. 79 | 3 | 14 | ditto | 1·5 | |
| 10 | F. 62 | 6 | 16 | ditto | 8 | Recovered swallowing. Gastrostomy allowed to close |
| 11 | F. 61 | 5 | 7 | ditto | Died | Emaciated woman. Pharyngeal carcinoma |
| 12 | F. 45 | 3 | 8 | ditto | Died | Hæmorrhage from œsophagus |
| 13 | F. 74 | 4 | 10 | ditto | 0·5 | Very emaciated |
| 14 | M. 77 | 8 | 10 | ditto | ? | Not traced. Immediate result satisfactory |
| 15 | M. 62 | 6 | 10 | Dilatation | 2 | Dilated three times |
| 16 | M. 68 | 8 | 16 | ditto | 5 | Dilated twice |
| 17 | M. 60 | 6 | 14 | Gastrostomy | 0·5 | |
| 18 | F. 61 | — | — | Nil | 0·5 | Refused examination, but diagnosis clinically certain |
| 19 | M. 41 | 6 | 10 | Gastrostomy | 2 | |
| 20 | M. 58 | 12 | 14 | ditto | Died | Gangrene of lung |
| 21 | M. 48 | 7 | 10 | Dilatation | 3 | |
| 22 | M. 61 | 24 | 12 | ditto | 1 | |
| 23 | M. 61 | 5 | 10 | ditto | Died | Mediastinitis. ? injury |
| 24 | M. 54 | 4 | 10 | ditto | 12 | Repeated dilatations |
| 25 | M. 69 | — | 12 | ditto | ? | Satisfactory on discharge. Not traced |
| *26 | M. 67 | 3 | 12 | Dilatation, intubation | 23 | **First case of intubation.* Intubated after 12 months' dilatation. Tube in place 11 months |
| 27 | M. 60 | 9 | 15 | Dilatation | 4·5 | |
| 28 | M. 59 | 2 | 10 | ditto | ? | Satisfactory on discharge. Not traced |
| 29 | M. 69 | — | 8 | Intubation | ? | Satisfactory on discharge. Not traced |
| 30 | M. 62 | 12 | 14 | ditto | Died | Extension growth involving lower 4 inches of œsophagus |
| 31 | M. 49 | 4 | 17 | ditto | 2 | Tube vomited next day. Replaced satisfactorily |
| 32 | M. 64 | 6 | 12 | ditto | 2 | Tube displaced in violent coughing 3 weeks later. Replaced satisfactorily |
| 33 | M. 64 | 4·5 | 13 | ditto | ? | Satisfactory on discharge. Not traced |
| 34 | M. 55 | 10 | 18 | ditto | ? | Tube vomited next day. Replaced satisfactorily |
| 35 | M. 54 | 2·5 | 14 | Intubation | Died | Emaciated man. ? injury to œsophagus |
| 36 | F. 66 | 10 | 11 | ditto | ? | Emaciated woman with dense stricture. Satisfactory, but not traced |
| 37 | M. 64 | 6 | 10 | ditto | 1·5 | Fungating mass of growth. Swallowed well on discharge |
| 38 | M. 66 | 3 | 16 | Dilatation, Intubation | 12 | Dilatation, followed 6 months later by intubation. Very satisfactory |
| 39 | M. 65 | 3·5 | 10 | Intubation | 5 | Died from pulmonary complications. Swallowed well to end |
| 40 | M. 69 | 2 | 11 | ditto | 18 | Passed tube but continued to swallow well. Section, squamous carcinoma |
| 41 | F. 76 | 4 | 14 | Nil | 0·5 | Emaciated woman. Impassable stricture. Really dying on admission |
| 42 | F. 58 | 8 | 10 | Intubation | 2 | Girdle pain first symptom. Sudden death. ? hæmorrhage |
| 43 | M. 71 | 3 | 10 | ditto | 2 | Passed tube but continued to swallow well |
| 44 | M. 57 | 8 | 11 | Gastrostomy | Died | Hæmorrhage from aorta, which was perforated by carcinoma |
| 45 | M. 52 | 4 | 22 | ditto | 1 | |

| | | | | | | |
|---|---|---|---|---|---|---|
| 40 | M. 69 | 2 | 14 | Nil | 0·5 | Emaciated woman. Impassable stricture. Really dying on admission |
| 41 | F. 76 | 4 | | | | |
| 42 | F. 58 | 8 | 10 | Intubation | 2 | Girdle pain first symptom. Sudden death. ? hæmorrhage |
| 43 | M. 71 | 3 | 10 | ditto | 2 | Passed tube but continued to swallow well |
| 44 | M. 57 | 8 | 11 | Gastrostomy | Died | Hæmorrhage from aorta, which was perforated by carcinoma |
| 45 | M. 53 | 4 | 10 | Intubation | 9 | Swallowing very satisfactory |
| 46 | M. 72 | 1 | 10 | ditto | Died | Large fungating carcinoma. Purulent infection of mediastinum |
| 47 | M. 62 | 3 | 10 | ditto | 3 | Swallowed well |
| 48 | M. 42 | 3·5 | 7 | Gastrostomy | 1+ | Impassable stricture at upper end of œsophagus. Not traced |
| 49 | F. 56 | — | 11 | Nil | 18+ | Old case cardiospasm. All signs of carcinomatous stricture, but no section |
| 50 | M. 60 | 1·5 | 11 | Intubation | Died | Large carcinomatous ulcer, possible perforated by tube |
| 51 | M. 69 | 2·5 | 10 | Intubation | 5 | Tight stricture, 1·5 inches long. *First use of aluminium dilators* |
| 52 | M. 47 | 3 | 15 | Nil | Died | Cardiac failure during anæsthesia. Post mortem, extensive carcinoma of lower end |
| 53 | F. 50 | 4 | 10 | Intubation | ? | Satisfactory on discharge. Not traced |
| 54 | F. 67 | 3 | 16 | Nil | ? | Malignant stricture seen, but impassable. Swallowing fairly. Died some months later |
| 55 | F. 42 | 9 | 7 | Gastrostomy | ? | Extensive carcinoma of pharynx and upper œsophagus |
| 56 | M. 54 | 11 | 8 | ditto | 2 | Extreme emaciation. Impassable stricture |
| 57 | M. 53 | 9 | 11 | Intubation | 1 | Large fungating mass. Tube inserted through *rubber cone* (*first use*). Swallowed well |
| 58 | M. 59 | 6 | 13 | ditto | 4·5 | Nodular growth. Tube with rubber cone |
| 59 | M. 57 | 3 | 9 | Dilatation | 3·5 | Nodular growth protruding from lateral wall. Too high for intubation. Sudden death. ? hæmorrhage |
| 60 | M. 56 | 5 | 10 | Intubation | 3 | Polypoid mass anterior wall. Sections, squamous carcinoma |
| 61 | M. 70 | 4 | 14 | ditto | Died | Carcinoma lower end with old perforation into subphrenic abscess |
| 62 | F. 60 | 3 | 10 | ditto | 8 | Fragile woman. Very successful |
| 63 | M. 70 | 12 | 12 | ditto | Died | Bronchopneumonia. Probably perforation of lung a week before intubation |
| 64 | F. 46 | 4 | 10 | ditto | 12 | At first very successful, but growth extended above tube, fungating in upper œsophagus |
| 65 | M. 78 | 10 | 16 | Dilatation | 2 | Tight constriction. Ulcerated ring of carcinoma |
| 66 | M. 53 | 4 | 10 | Intubation | 2 | Swallowed well, but troublesome cough. Probably fistula before operation |
| 67 | M. 75 | 6 | 13 | ditto | 0·5 | At first satisfactory, then intractable vomiting. Extension to stomach |
| 68 | M. 62 | 6 | 10 | ditto | 0·5 | Swallowed well, but large amount of pus vomited indicated abscess of some standing |
| 69 | M. 72 | 2·5 | 8 | Nil | Died | Soft mass of ulcerated growth. Probably perforation |
| 70 | M. 42 | 2·5 | 10 | Dilatation | 2 | Stricture with no apparent ulceration |
| 71 | M. 66 | 2 | 10 | Gastrostomy | ? | Large lateral nodule projecting into œsophagus |
| 72 | F. 50 | 6 | 10 | Intubation | 9 | Very soft fungating carcinoma |
| 73 | M. 81 | — | — | Nil | — | Swallows fluids. Too old for investigation |
| 74 | M. 52 | 1 | 16 | Gastrostomy | 7+ | Hard tumour bulging wall on right. Possibly extra-œsophageal |
| 75 | M. 61 | 3 | 10 | Intubation | 1 | Swallowed well on discharge |
| 76 | M. 61 | 3 | 13 | Nil | 4 | Attempts failed owing to severe hæmorrhage from fungating growth |
| 77 | M. 72 | 2·5 | 10 | Intubation | 4 | Fungating growth beyond tight stricture. Tube passed post mortem |
| 78 | M. 71 | 4 | 10 | ditto | — | Dense ring carcinoma. Flanged tube very successful |
| 79 | M. 70 | 3 | 10 | ditto | 3 | Dense ring of fungating carcinoma. Very successful |
| 80 | M. 56 | 3 | 10 | ditto | — | Ulcerated ring of carcinoma |
| 81 | M. 61 | 3 | 9 | ditto | Died | Temporary success, but gangrene of lung (noted at operation) |
| 82 | M. 72 | 5 | 10 | ditto | 1 + | Immediate success complete. Passed tube post mortem, but continued to swallow well |
| 83 | M. 55 | 3 | 16 | ditto | 2·5 | Tube entered stomach, but remained in position. Swallowed well |
| 84 | M. 59 | 4 | 10 | ditto | — | Ulcerated growths. Satisfactory |

*Continued on next page*

*Table IV.*—Treatment and Results of 100 Cases of Œsophageal Carcinoma—*continued.*

| Number | Sex and Age | Duration (Months) | Site (Inches from Teeth) | Treatment | Survival (Months) | Remarks |
|---|---|---|---|---|---|---|
| 85 | M. 64 | 5 | 14 | Intubation | — | One month before, gastrostomy elsewhere. Swallowed comfortably after intubation |
| 86 | M. 70 | 12 | 15 | ditto | — | Ulcerated growth 3 inches long |
| 87 | M. 47 | 2 | 8 | ditto | — | Stricture dilated with great difficulty. Tube caused severe irritation. Swallowed well after removal |
| 88 | F. 50 | 2 | 10 | ditto | 5 + | Very soft growth. Squamous carcinoma |
| 89 | M. 50 | 12 | 13 | ditto | — | Swallowed well on discharge. Ulcerating carcinoma |
| 90 | M. 72 | 8 | 11 | ditto | 2·5 | Swallowed nothing on admission, comfortably on discharge. Complete circle of fungating carcinoma |
| 91 | M. 54 | 7 | 8 | Dilatation | — | Improved. Too high for intubation |
| 92 | M. 69 | 3 | 12 | Nil | — | Impassable stricture, but could swallow fluids |
| 93 | M. 54 | 2 | 10 | Dilatation | — | Soft fungating carcinoma, unsuitable for intubation |
| 94 | M. 59 | 3 | 12 | Intubation | — | Dense stricture. Swallowed well on discharge |
| 95 | M. 73 | 6 | 14 | ditto | — | Tight stricture 3 inches long. Sudden onset. Swallowed well on discharge |
| 96 | F. 87 | 1 | 8 | Nil | — | Impassable stricture |
| 97 | M. 68 | 38 | 7 | Gastrostomy | 10 | Large impenetrable mass. Paralysis both cords. Severe respiratory obstruction |
| 98 | M. 71 | 2 | 12 | Intubation | — | Dense stricture. Swallowing very satisfactory on discharge |
| 99 | M. 61 | 2 | 12 | Nil | — | Large soft fungating carcinoma. Impenetrable, but swallowed fairly |
| 100 | M. 65 | 6 | 10 | Dilatation | — | Soft fungating growth. Unsuitable for intubation. Swallowing improved |

# AN OPERATION FOR SLINGING A DROPPED SHOULDER.*

By ARNOLD K. HENRY,
PROFESSOR OF CLINICAL SURGERY AT THE ROYAL SCHOOL OF MEDICINE, CAIRO.

It is often well for the surgeon dealing with nerve lesions to have an orthopædic string to his neurological bow. Especially is this the case in a hospital practice where the almost immediate restitution of function is confidently expected by the patient as a result of operation. Nerve suture in this circumstance, with its tedious interlude and uncertain promise, is not always the chosen procedure, and the following is an example of a short cut to function which I believe has not been previously described.

History.—The patient, a well-developed youth of 17, was referred to me from the Medical Division of Kasr el Aini Hospital by Dr. Yussef Barrada. He had been operated on elsewhere eighteen months before for enlarged glands in the left side of the neck, and he was discharged in a fortnight, as he said himself, "perfectly satisfied". He was in the habit of lifting weights and doing physical exercises, and two months later he felt difficulty in using his left arm. After a few days he noticed that his shoulder-blade was projecting upwards, and that it was sliding to and fro more freely than he thought it should. He also felt a painful drag on his left shoulder. He then consulted a doctor, and was advised to bandage his arm with the elbow supported. This was done for a short period without relief.

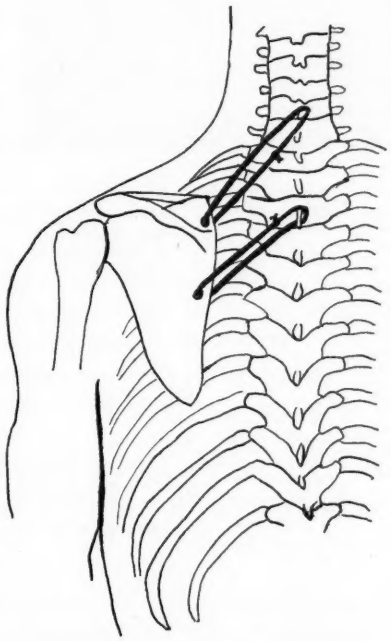

Fig. 63.—The scapula is slung to the spines of the 6th cervical and 3rd dorsal vertebræ by two strips of fascia.

On Admission.—There was a 2½-in. scar of operation at the upper third of the left sternomastoid, with some induration deep to the scar. The point of the left shoulder was depressed, and the medial angle of the scapula projected prominently at the left side of the neck. The whole trapezius muscle was wasted and did not contract. The sternomastoid was paretic. There was slight winging of the left scapula which the patient could correct.

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* From the Surgical Unit, Kasr el Aini Hospital, Cairo. Shown at a meeting of the Egyptian Branch of the British Medical Association, Dec. 16, 1926.

*Movements.*—The patient was unable: (1) To raise his left arm above the horizontal position; (2) To touch the right cheek with his left hand; (3) To put his left hand freely behind his back.

*Sensation.*—There was blunting of sensations of touch and pain over the idstribution of the great auricular and the transverse cutaneous cervical nerves.

Operation (*Figs.* 63, 64).—

*Incisions.*—Under a general anæsthetic, with the patient lying on his right side and his left arm raised above his head, incisions were made to expose: (1) The medial angle, and (2) The vertebral border of the scapula; (3) The 5th, 6th, and 7th cervical spines, and (4) The 1st, 2nd, and 3rd dorsal spines. Through incisions (1) and (2) the scapula was exposed sufficiently to allow two holes, each $\frac{1}{4}$ in. in diameter, to be bored through the bone $\frac{3}{4}$ in.

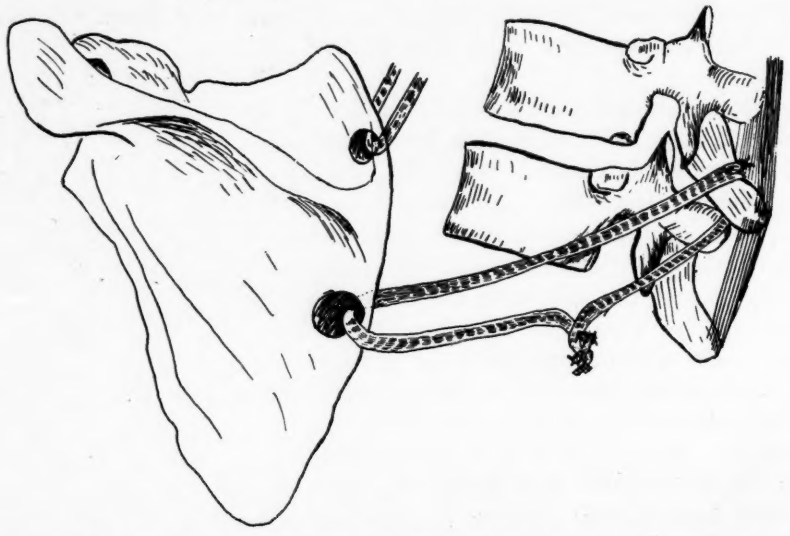

Fig. 64.—The fascial sling is passed through a hole in the scapula and round the base of the spinous process. Its ends are intertwined and sutured, and it is stitched to the interspinous ligaments.

from its margin, one just above the spine, the second $2\frac{1}{2}$ in. below it. Through incisions (3) and (4) the spines of the 6th cervical and 3rd dorsal vertebræ were exposed and their bases stripped of periosteum, care being taken to preserve the interspinous ligaments joining their distal ends.

*The Slings.*—While these exposures were in progress on the patient's back, a strip of fascia lata 10 in. long and 1 in. wide was outlined through a 12-in. incision on the outer side of the thigh by two longitudinal cuts. The strip was left *in situ* till the work on the back was sufficiently advanced; it was then halved longitudinally, one half being removed and threaded by an end through the hole in the medial angle of the scapula. The other end was passed round the spine of the 6th cervical vertebra *deep* to the interspinous ligaments joining that vertebra to its neighbours. The sling was then drawn as tight as possible and its two ends were intertwined and sutured together with No. 3 catgut. In addition, the loop of the sling which embraced the vertebral

spine was fixed by sutures to the interspinous ligaments. The remaining half of the fascial strip was now detached from the thigh and the procedure was repeated in such a way as to link the second hole in the scapula with the 2nd dorsal spine.

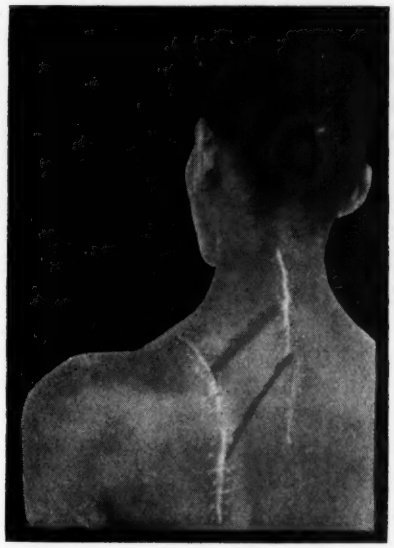

FIG. 65.—The patient after operation with his arm by his side. (The skin is chalked to show the positions of the two slings.)

After the closure of the wounds in two layers the left wrist was fixed with a plaster bandage to the head, with the palm of the hand resting on the vertex. There was much difficulty in persuading the patient of the necessity for this posture, and the head bandage had to be supplemented by a plaster support extending from the trunk along the upper limb. He broke this twice, and when, eighteen days after his operation, I saw that he had succeeded in removing the entire fixture, I was prepared to find that our efforts had been defeated. The patient, however, had been testing the operative results for himself, and he was satisfied that there was now no pain in his shoulder, that he could raise his left arm freely above his head, and put it easily behind his back. After some persuasion he consented to remain with his arm bandaged to his head for ten more days, and in return I agreed that the limb should be set free each night when he had lain down.

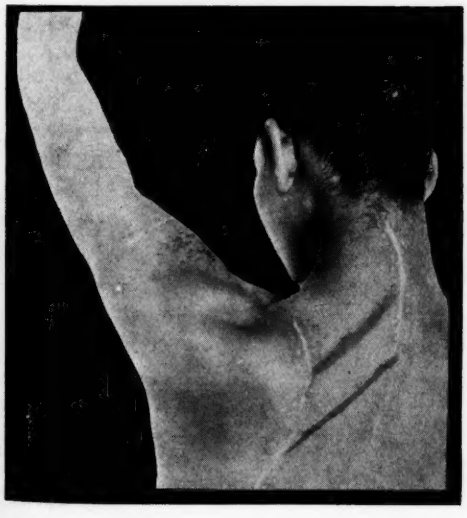

FIG. 66.—The patient with his arm elevated.

Three months after operation I had the opportunity of showing the patient to the Egyptian Branch of the British Medical Association. The shoulder was painless, movements of the left upper limb were free and complete (*Figs.* 65, 66). The patient unconsciously illustrated this by pulling off his shirt over his head. There was bony thickening at the points where the fascial slings passed through the scapula, and the slings themselves could be felt under the wasted trapezius as thick cords which stood out and became tense when the arm was raised above the head. During this movement, after the deltoid had brought the arm to the horizontal position, the slings steadied and supported the scapula in such a way as to allow the serratus magnus (*serratus anterior*, B.N.A.) to bring

the lower angle of the scapula forwards and outwards and so complete the elevation of the arm to the vertical position.

## SUMMARY.

A method is described by which a case of shoulder-drop was successfully treated by slinging the scapula to the spines of the 6th cervical and 3rd dorsal vertebræ by means of 10 in. by ½ in. fascial strips taken from the thigh.

My thanks are due to Drs. Handousa and Hafez for their assistance at the operation, to Dr. Shafik Abd el Malek for his careful notes of the case, and to Dr. Boris Boulgakow for his diagrams. The photographs were most kindly taken by Prof. R. Heathcote.

# POLYPI OF THE INTESTINE: WITH SPECIAL REFERENCE TO THE ADENOMATA.*

By JAMES HAROLD SAINT, Newcastle-upon-Tyne.

Some confusion seems to exist in the literature as to the exact meaning of the term 'polypus of the intestine', some authors being inclined to regard it as designating a particular type of tumour, which, however, is never well differentiated from adenoma or inflammatory hyperplasia; but on the whole it would seem that it is more commonly, and rightly, used in a broader sense as the general term for any pedunculated or sessile growth projecting into the lumen of the bowel, either the result of hypertrophy or hyperplasia of the mucous membrane, or else a benign, true tumour. It is the latter conception of the term which the writer accepts and applies in this paper.

The intestinal wall is made up of various types of tissue, viz., the mucous membrane, areolar tissue forming the submucous and subperitoneal layers, the unstriped fibres of the musculature, vascular tissue, and the outermost endothelial peritoneal coat. From these various tissues tumours of different varieties may arise, and, enlarging, project into the lumen of the bowel to form polypi. Thus it is possible to find adenomata and papillomata arising from the mucous membrane; fibromata, lipomata, and myxomata from the areolar tissue (though only very rarely from that of the subperitoneal layer); myomata from the musculature; and hæmangiomata from the vessels. The peritoneal coat alone appears innocent of any attempt at tumour formation. Thus the term polypus covers a heterogeneous collection of tumours, and it is necessary in using it to be specific from the histological standpoint.

This work was undertaken in order to study the pathology of the various types of polypi met with in the intestine, the relative frequency of their occurrence, and their favourite sites of predilection. The specimens were obtained from autopsies, and represent a series collected at the Mayo Clinic over a period of several years.

Polypi have been found in all parts of the intestine, but are met with in certain positions more often than in others. They may be single or multiple, and vary very much in size. In 13 of the cases the polypi were met with in the small intestine, the remaining 44 being found in the large bowel.

## POLYPI OF THE SMALL INTESTINE.

The following tables show the distribution and types of the polypi met with in the small intestine in this series of cases.

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* This work was carried out under the direction of Dr. H. E. Robertson of the Section on Pathologic Anatomy, Mayo Clinic, Rochester, Minnesota, U.S.A., the writer being a Fellow in Surgery on the Mayo Foundation.

*Table I.*—Distribution.

| | |
|---|---|
| Duodenum .. .. | 1 |
| Jejunum .. .. .. | 2 |
| Ileum .. .. .. | 10 |

*Table II.*—Types of Polypi.

| | |
|---|---|
| Glandular hyperplasia .. | 9 |
| Hæmangioma .. .. | 1 |
| Globocellularis .. .. | 1 |
| Lipoma .. .. .. | 1 |
| Adenoma .. .. .. | 1 |

**Glandular Hyperplasia.**—From these tables it is seen that the ileum is the favourite site for polypi in the small intestine, and that, as regards type,

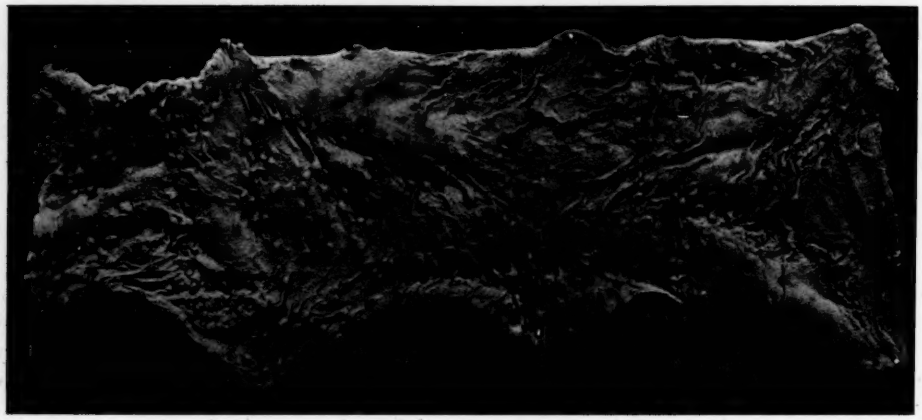

Fig. 67.—Multiple polypi of small intestine. Glandular hyperplasia.

those formed by glandular hyperplasia easily outnumber the other varieties. This type takes the form of multiple excrescences on an average about 1 mm. in diameter, arising from the mucous membrane (*Fig.* 67). They may be either sessile or pedunculated, in the latter case the pedicles being usually not more than 1 mm. or 2 mm. in length, and most of them are seen to arise from the crests of the valvulæ conniventes. On microscopic examination the polypoid appearance is seen to be due to a rising up of this mucous membrane and submucosa above the general level on to the lumen of the intestine (*Fig.* 68). The villi and glandular follicles between them are similar in appearance to those between the excrescences, so that the condition is apparently merely one of local hyperplasia. In a few instances, forming the pedicle of the polypus, is a mass of lymphoid tissue. This has a fine connective-tissue stroma derived from the branching of that of the pedicle, and a few thin-walled capillaries can also be seen. It would seem that these polypi are formed by a hyperplasia of some of the lymphoid follicles in the intestinal mucosa, as these ordinarily do not project into the lumen of the bowel to such a marked degree. Kanthack and Furnival in

Fig. 68.—Section of specimen shown in *Fig.* 67. (× 18.)

their case found similar glandular hyperplasia with normal lymphoid follicles occurring chiefly in the duodenum and jejunum; but in the present series 8 out of 9 cases were found in the ileum and the remaining 1 in the jejunum.

**Hæmangiomata.**—Hæmangiomata of the intestinal wall are rare, though more common in the small than in the large intestine. MacCallum in 1906 published a case of multiple cavernous hæmangiomata occurring throughout the length of the small intestine, but especially in its upper portion. At that time he was able to find only four other recorded cases, two of which were single and two multiple. The present case conformed to the most common macroscopic characteristics in that the hæmangiomata were small and multiple. They were found only in the jejunum. The larger tumours, about ½ cm. in diameter, pushing the mucous membrane before them, appeared as reddish nodules in the lumen of the intestine, and could also be seen shining through the peritoneal coat as dark-purplish patches. By transmitted light, a large number of these dark areas, varying in size from ½ cm. to 1 mm., were seen situated on the course of veins. At first the nodules were thought to be

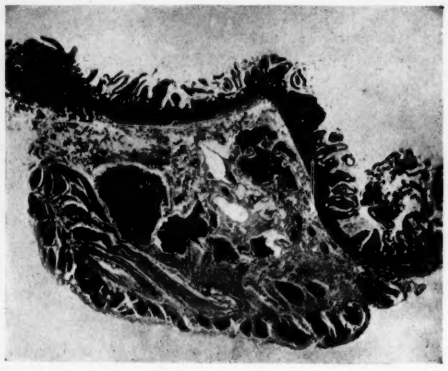

FIG. 69.—Section through hæmangioma of small intestine. (× 8.)

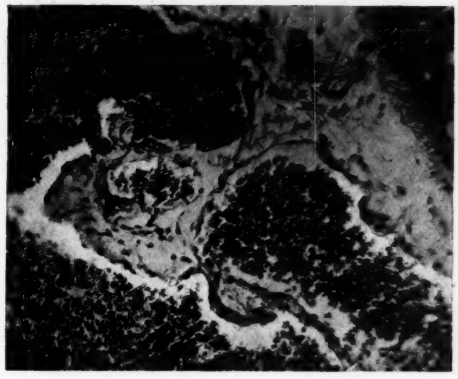

FIG. 70.—Hæmangioma of small intestine, showing endothelial lining of blood sinuses. (× 70.)

simple hæmorrhages, until microscopic examination proved them otherwise. A section through one of them (*Fig.* 69) shows that it is not a hæmorrhage but a vascular tumour. The submucous layer is greatly increased in thickness, and in it are seen numerous irregular, sinus-like spaces, each of which is lined by a definite layer of endothelium (*Fig.* 70) and contains blood in greater or less degree. External to the endothelium is the connective tissue of the submucosa, forming partitions between the sinuses, there being no suggestion of vessel-wall formation. In some of the sections the partitions between the blood sinuses are seen to end abruptly in the centre of a space, showing that the sinuses communicate freely with one another, thus proving the cavernous character of the tumour. There was no attempt at encapsulation.

Bennecke described an interesting case in which the cavernous tumours existed throughout the œsophagus and stomach as well as the intestine, and he considers them to resemble the cavernous hæmangiomata of the liver in their general character.

**Globocellularis.**—Another rare type of tumour found in this series,

forming a small polypus in the ileum, is the globocellularis, or carcinoid tumour, as it is sometimes called. It is also referred to as a primary carcinoma. Apparently the two usual sites for this type of tumour are the small intestine and the appendix.

Two of the best contributions to the literature on this subject are those of Bunting, and Rolleston and Jones. Bunting describes a case of "multiple primary carcinomata of the ileum" in which several small nodular structures were scattered throughout the upper part of the ileum. At the same time he quotes the only other six recorded cases he was able to find. All were multiple. He describes their characteristics as the multiplicity of primary foci occurring at other than the usual sites of occurrence of intestinal growths, the undifferentiated nature of the cells, their slight invasive tendency, and apparently slow growth. Krompecher was the first to draw attention to the resemblance between these tumours and the slowly growing basal-celled type of epithelioma.

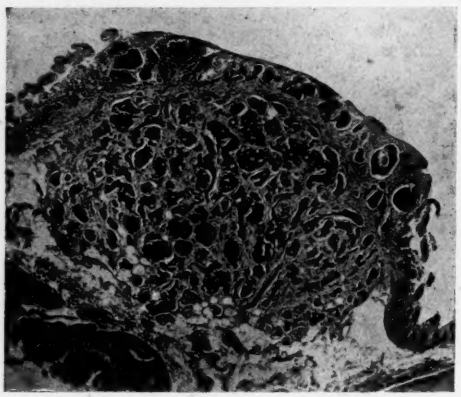

FIG. 71.—Carcinoid tumour (globocellularis) of ileum. (× 15.)

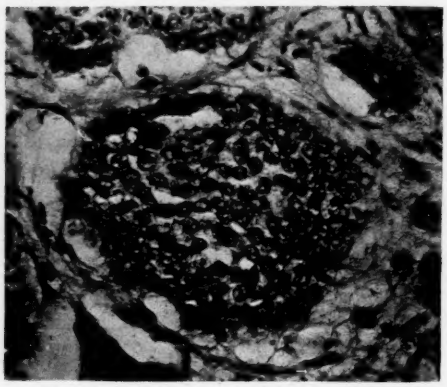

FIG. 72.—Higher magnification of *Fig.* 71, showing large nuclei and prominent nucleoli. (× 240.)

The 'primary carcinoma' of the appendix described by Rolleston and Jones has macro- and microscopic characteristics similar to these multiple tumours described by Bunting and to the specimen found in this series.

The tumour under review was single, in contrast to Bunting's, sessile, and about 4 mm. in diameter. To the naked eye it looked rather like an adenoma, but was paler in colour. Upon histological examination there were seen numerous nests of closely-packed cells in the submucosa, causing considerable thickening of that layer of the intestine at this point (*Figs.* 71, 72.) Internal to the cell-nests neither villi nor crypts of Lieberkühn were seen, their place being taken by gland-like spaces similar to the cell-nests of the tumour. The latter were varied in size and shape, and were separated from one another by a fairly dense connective-tissue stroma containing in its inner part some smooth muscle fibres derived from the muscularis mucosæ and in which only a few cells were seen. Occasionally cell-nests were seen to be connected with one another by slender columns of cells. The tumour-cells were small and uniform in size, round or almost so in shape, with scanty clear protoplasm, and possessed large vesicular nuclei staining well with basic

dyes and containing prominent nucleoli. Mitotic figures are described as being of rare occurrence, and none was observed in this case. In some of the cell-nests a tendency to the formation of a lumen was seen, the appearance being due to the degeneration of the centre cells. The tumour, though not encapsulated, was limited to the submucosa and exhibited no tendency to penetrate the muscular coat. This type of tumour has given rise to much controversy, and whether or not it can be accurately designated carcinoma seems to be still a debatable point.

**Lipomata.**—Although lipomata may arise from the submucous coat and grow inwards, or from the subperitoneal coat and project outwards, the

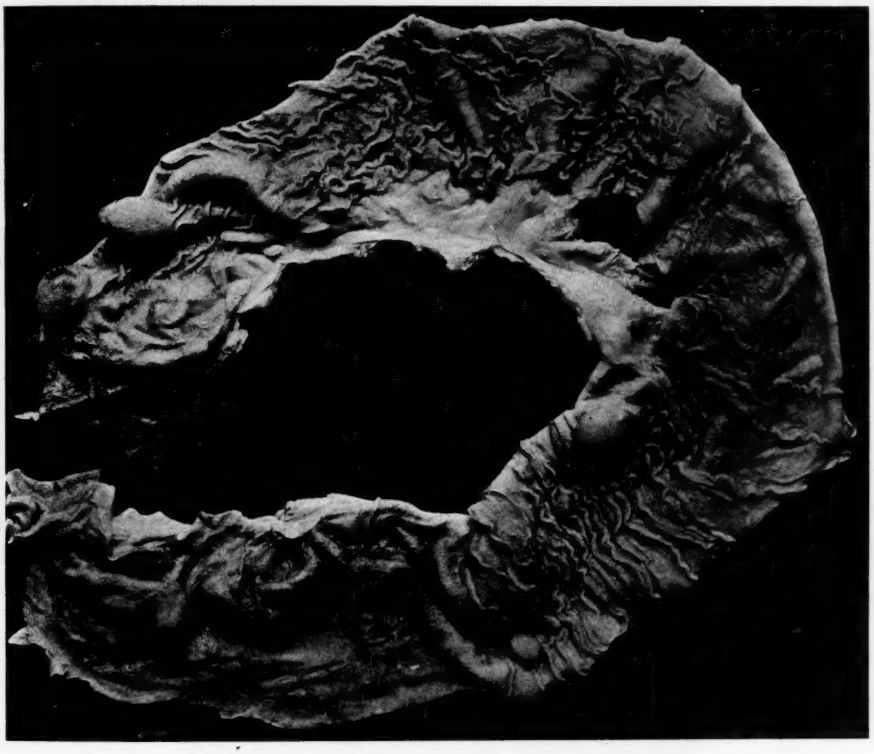

FIG. 73.—Multiple submucous lipomata of ileum.

former is the more common as a site of occurrence of these tumours. Lipomata may occur anywhere in the intestinal tract, may be single or multiple, sessile or pedunculated. Perhaps their only claim to any importance is the large size to which they may grow, finally obstructing the intestine or being instrumental in bringing about the condition of intussusception.

In a case of this series, multiple lipomata were found in the ileum (*Fig.* 73). They were six in number, and varied in size from 1·5 cm. to 0·5 cm. in diameter. All of them were sessile, but one or two were slightly constricted at their bases, indicating the commencement of a pedicle. The mucous membrane covering them appeared tense and smooth, and the fatty tissue shining through it gave to the tumours a yellowish tinge. Microscopically one of these tumours is seen to consist of a circumscribed mass of fat-cells

lying in the submucosa, resting on the muscular coat, and covered by mucous membrane in no wise differing from that lining the bowel.

**Adenomatous Polypi.**—These are of much less common occurrence in the small than in the large intestine. In the former they are most often met

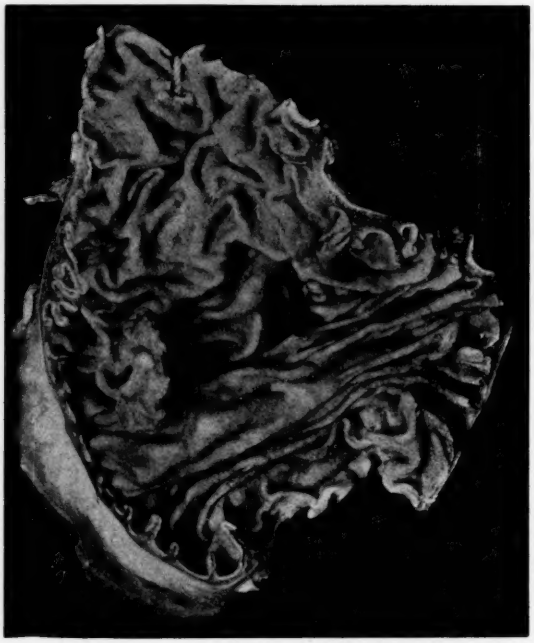

FIG. 74.—Adenoma of duodenum: probe in ampulla of Vater.

with in the duodenum. It was in this part of the small gut that the only one in this series was found (*Figs.* 74, 75). The polypus was situated about 2 cm. below the duodenal papilla; it measured 1·5 cm. in length, and had a

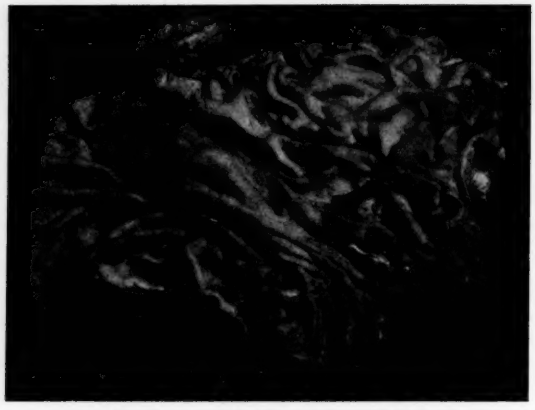

FIG. 75.—Another view of *Fig.* 74.

diameter of about 0·5 cm. It was pedunculated, its pedicle being 0·5 cm. in length. On microscopic examination the polypus proved to be a typical adenoma, the morphological characteristics of which will be described at some length when polypi of the large intestine come under discussion later.

The following tables show, first, the relation between number and sex, and, secondly, the age incidence, of the cases encountered in this series.

*Table III.*—Number and Sex Incidence.

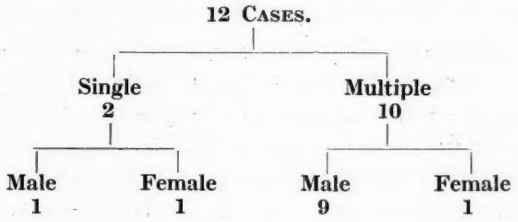


*Table IV.*—Age and Sex Incidence.

| Age | Male | | Female | |
|---|---|---|---|---|
| Years | Single | Multiple | Single | Multiple |
| 10–19 | 0 | 1 | 0 | 0 |
| 20–29 | 0 | 3 | 0 | 0 |
| 30–39 | 0 | 1 | 0 | 0 |
| 40–49 | 0 | 2 | 0 | 0 |
| 50–59 | 1 | 1 | 1 | 0 |
| 60–69 | 0 | 1 | 0 | 1 |

From *Table III* it is seen that in the small intestine multiple polypi were of much more frequent occurrence than those which are single.

*Table IV* presents no outstanding facts from which any conclusions can be drawn. It is interesting that while the first case of multiple polypi (glandular hyperplasia) tabulated above was in a boy of 16, the last was in a woman of 63, showing that the condition can occur at widely differing ages; but whether or not the condition begins early and persists throughout life it is not possible to say. Kanthack and Furnival's specimen was found at autopsy in a girl of 13.

## POLYPI OF THE LARGE INTESTINE.

Polypi occur much more frequently in the large than in the small intestine, and may be classified into two groups, namely: (1) Those resulting from inflammatory or ulcerative processes in the bowel; (2) True tumours, of which the only representatives in this series of cases were adenomata and lipomata. The following tables show the types of polypi and their distribution in the various parts of the large bowel:—

*Table V.*—Distribution.

| | |
|---|---|
| Cæcum | 8 |
| Ascending colon | 6 |
| Transverse colon | 3 |
| Descending colon | 5 |
| Sigmoid colon | 20 |
| Rectum | 5 |
| Part of colon unspecified | 4 |

*Table VI.*—Types of Polypi.

| | |
|---|---|
| Inflammatory | 6 |
| Lipoma | 2 |
| Adenoma | 36 |

Thus the sigmoid was the most frequent site of occurrence of polypi in the large bowel, and adenomata were the type of polypus by far the most frequently met with.

### POLYPI ASSOCIATED WITH ULCERATIVE COLITIS.

These are not true tumours, but consist of strips of mucous membrane which have become detached along almost their whole length due to the undermining character of the ulceration. Their number is probably greater in the milder forms of ulceration, since if this is very severe one can imagine a widespread detachment of the mucous membrane, leaving behind only enough to form comparatively few polypi. In the six cases in this series the colitis was in an advanced stage, with thickening and contraction of the colon wall, disappearance of the haustra, and marked denudation of the mucosa, so that the resulting polypi were few in number. In size these polypi vary considerably, but most of them are long and thin, showing little alteration in diameter between their free and attached ends. Microscopically, glandular and connective tissue is seen; the glands have the same appearance as those of the normal mucous membrane, but their relationships have been disturbed, doubtless as a result of the ulcerative process, so that they appear scattered throughout the mass of inflammatory tissue. The connective tissue is considerable in amount, and is heavily infiltrated with chronic inflammatory cells—lymphocytes, plasma cells, and mast cells (*Fig.* 76).

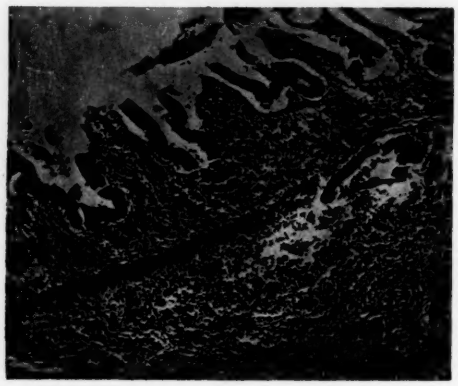

FIG. 76.—Section of inflammatory polypus: dense cellular infiltration of connective tissue. (× 36.)

### LIPOMATA.

Lipomata in the large intestine have the same naked-eye and microscopic characteristics as those found in the small bowel. In the first of the two cases of lipomata in this series the tumour was sessile, situated in the cæcum, was about 1½ cm. in diameter, and protruded into the lumen for 3 or 4 mm. The lipoma in the second case was a sessile tumour 2 cm. in diameter and about 1 cm. in depth, adjacent to the posterior segment of the ileocæcal valve, while in the cæcum three or four small sessile adenomata were present. Lewisohn published an interesting case in which a patient had been suffering from intermittent attacks of intestinal obstruction of short duration and right-sided abdominal pain. At operation a pedunculated lipoma the size of a plum, originating from the inner wall of the ascending colon, and gangrenous in its distal part, was found. Lewisohn thought that the gangrene had evidently been caused by the tumour having been caught in and constricted by the ileocæcal valve at frequent intervals.

### ADENOMATA.

The adenomata form a very interesting group of polypi. They may be described as tumours arising from the glandular epithelium in which the epithelium and connective tissue grow co-ordinately. They form the most common type of polypus met with in the large intestine, and as such have interested pathologists for many years, so that the literature concerning them has assumed large proportions. They vary very much in size, and may be single or multiple. In this series their size varied from that of a pinhead up to a diameter of several centimetres. The larger type of adenoma is usually single.

Multiple adenomata may be divided into two distinct types :—

1. The entire colon or a limited portion of it is covered with small pedunculated and sessile polypi in such numbers that the mucosa is almost hidden. This is the type referred to as 'multiple polyposis of the colon' or 'colitis polyposa' by such authors as Virchow, Luschka, Doering, Struthers, and others quoted by them. It would seem that this condition is always associated with an inflammatory or ulcerative process in the colon. Judging from the histological reports in the literature accompanying this type, it is of interest, however, to notice that true adenomatous formation does not necessarily predominate, but that many of the polypi may show only simple glandular hyperplasia. Dilatation of the glands into cyst-like spaces has been observed (Virchow, Luschka, Libert), and to this condition Virchow gave the name 'colitis polyposa cystica'. It is probably an advanced stage of colitis polyposa, and is due to the occlusion of the mouths of secreting glands by the fibroblasts formed during the inflammatory process. This condition was not met with among the cases of the present series, but is mentioned to avoid confusion with the second type, which is also sometimes referred to as 'multiple polyposis'.

FIG. 77.—Pedunculated adenoma in section. (× 8.)

2. In this type, which is perhaps best described under the name 'multiple adenomata', the mucosa is studded with these tumours, which, however, are few in number when compared with the myriads found in the first type. They may be either sessile or pedunculated, may grow to a fair size, but are usually not so large as the single adenomata. They are usually seen growing on the free edges of folds of the mucous membrane.

Given below is a detailed description of a typical pedunculated adenoma with a horizontal diameter of 2 mm. and measuring 2·5 mm. proximo-distally, mounted on a pedicle about 1·5 mm. in length (*Fig.* 77).

The tumour is darker in colour than the mucous membrane of the bowel, and its surface is smooth. On small magnification the mouths of the glands

opening on the surface are seen. The pedicle is smooth and covered with mucous membrane continuous with that of the intestine. It is somewhat cylindrical in shape, but is broader at its base where it is attached to the intestinal wall, its diameter decreasing until the point is reached from which the tumour is appended. On section, the pedicle is seen to consist of a core of connective tissue growing up from the submucosa. On reaching the tumour the core loses its identity by dividing into processes which pass in among the glandular tissue. Microscopically the tumour is seen to consist of glandular and connective tissue, the latter acting as the supporting framework of the former.

The glandular tissue consists of long tubular glands mostly perpendicular to, and opening on, the surface of the tumour. These glands are lined by columnar epithelium. In the neighbourhood of the pedicle the glands resemble those of the normal intestinal mucosa, i.e., they are about the same in diameter and are lined by cells each having a single oval nucleus at its base. The longitudinal axis of the nucleus is parallel to that of the cell. Very little or no cytoplasm is visible, the cells being distended with mucus, and some of this substance is also lying free in the lumen of the gland. Towards the periphery of the tumour the glands change somewhat in character. They enlarge considerably both in length and diameter, and the lining epithelium assumes a different complexion, the cytoplasm having a granular appearance. However, many of the cells are distended with mucus, this clear material standing out prominently in contrast to the granular cytoplasm of adjacent cells. The nuclei are arranged in three or four layers, and some of them occupy a position in the inner part of the cell. In other words, these cells have lost their polarity. Here and there regular mitotic figures are seen in the inner parts of the cell. There are some thickenings of the epithelium appearing as vegetations projecting into the lumina of the glands. These changes are marked with increasing distinctness the more nearly the glands are situated to the periphery of the tumour. It would seem from these changes that the cells of the glands near the pedicle, like those of the mucous membrane, are concerned entirely with the secretion of mucus, whereas those towards the periphery are apparently beginning to be concerned with proliferation, although still retaining their mucus-secreting power.

The interglandular connective tissue consists of branches from that of the pedicle ramifying in the interstices between the glands and forming the supports on which they rest. Towards the periphery the connective tissue forms a fine reticular network, in the meshes of which vessels and cells are seen. The vessels are capillaries which for the most part are filled with red cells. The cells in the network are numerous, and various types are seen: (1) Elongated connective-tissue cells with spindle-shaped nuclei. The nearer the pedicle the greater is the tendency of these cells to be closely parallel to one another and so form fasciculi. Towards the periphery their numbers diminish considerably. (2) Small round lymphocytes in abundance, being grouped together in various places. (3) Large mononuclear cells with a pale clear protoplasm and a darker staining nucleus which has a reticulated structure. These are not nearly so numerous as the small lymphocytes. (4) Plasma cells—large protoplasmic cells, each with a round eccentric nucleus

having in it several chromatin granules arranged mostly round the periphery, giving it the appearance of a spoked wheel. (5) Eosinophilic leucocytes. (6) Mast cells with a small nucleus and a cytoplasm which contains large numbers of granules staining deeply with basic dyes. (7) Red blood-cells, present in large numbers in the interstices of the connective tissue.

The pedicle is seen to take origin from the loose areolar submucosa. It is composed of strands of connective tissue running up into the tumour, encased in mucous membrane continuous with that of the intestine. The connective tissue consists of long parallel fibrils which in the centre of the pedicle are densely packed together, while towards the periphery they are more loosely arranged. The muscle fibres of the muscularis mucosæ are

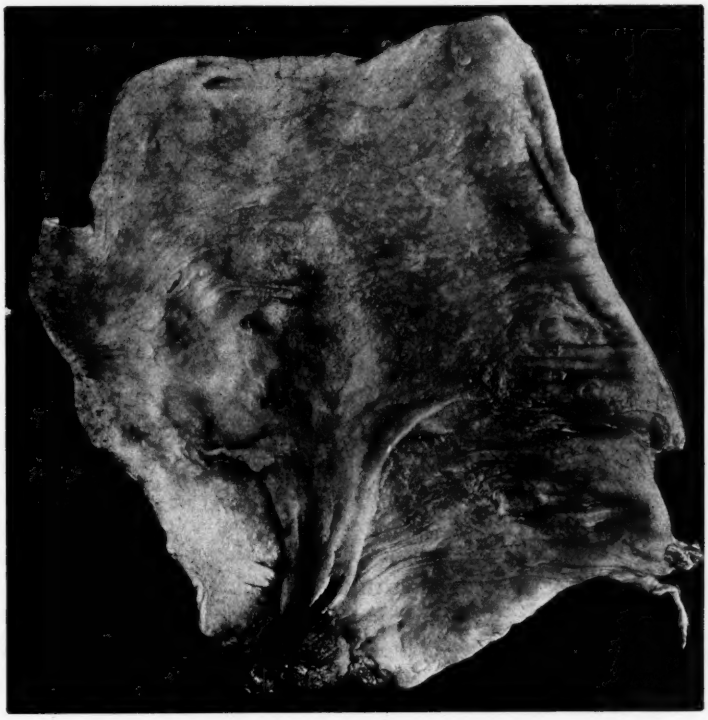

Fig. 78.—Large pedunculated adenoma of colon.

continued upwards into the pedicle immediately subjacent to the mucous membrane, but as they proceed distally the fibres become attenuated and finally disappear. There are numerous blood-vessels, branches from those in the submucosa running upwards in the pedicle to supply the tumour. They are of small size, and their walls consist of lining endothelium surrounded by a single layer of smooth muscle fibres. Compared with the connective tissue between the glands of the tumour, that of the pedicle is much less cellular. Particularly is this so in the centre of the pedicle, only a few spindle-shaped nuclei being present in this situation. Proceeding towards the periphery of the pedicle more cells are seen, the most abundant being the small round lymphocytes, although the other types of cells mentioned when discussing the interglandular connective tissue make their appearance.

The mucous membrane covering the pedicle is essentially the same as that of the intestine with which it is continuous. The glands are the same size, and are lined by clear, mucus-laden columnar cells. Between the glands is the supporting reticulated connective tissue continuous with the periphery of that of the pedicle.

As an adenoma increases in size it undergoes changes which are of considerable importance and significance.

Macroscopically one sees a more or less spherical pedunculated tumour, dark red in colour, and about the size of a hazel nut (*Fig.* 78). The surface is velvety in appearance, distinctly lobulated, and covered with mucus in the recent state. Between the lobulations are clefts which extend inwards for a varying depth. The tissue is very soft and friable, and is easily damaged if not carefully handled.

The pedicle is of varying length, and has lost the rounded outline common to those which measure only a few millimetres. It is flattened—usually from above downwards—and is much broader at its base than at its distal end where it is attached to the tumour. Often it has a ribbon-like appearance produced by longitudinal thickenings of the connective-tissue core. It is smooth and of the same pale-red colour as the mucosa. Usually it arises from the summit of a fold of the mucous membrane of the bowel.

FIG. 79.—Cystic formation in an adenoma. (× 36.)

On section of such a polypus, well-formed vessels are seen proceeding upwards in the pedicle to supply the tumour. The connective-tissue core of the pedicle on reaching the adenoma can be seen dividing up into numerous branches spreading amongst the glandular substance, which is of a red colour and granular in appearance.

Histological examination of the adenoma shows that the glands near the pedicle are lined by regular columnar mucus-laden cells, each with a single large oval nucleus at its base. Little or no cytoplasm is seen. There is a distinct basement membrane composed of a single layer of more or less fused flattened cells of connective-tissue nature united edge to edge.

Proceeding outwards towards the periphery one first comes upon glands which exhibit characteristics similar to those found at the periphery of a small adenoma. Thus the glands are larger in diameter, the nuclei have proliferated, epithelial vegetations are seen, the protoplasm in many cells has become granular, and some cells have lost their polarity. Some of them are seen to have become cystic (*Fig.* 79). Nearer the periphery the glands undergo still further changes. In addition to being larger they become markedly irregular in contour, and are often segmented in the direction of their length. However, amongst these are seen glands which are only enlarged and have no irregularities of outline. The epithelial vegetations are larger,

the nuclei are more numerous and are also enlarged. They are slightly more hyperchromatic than those of the cells lining the glands near the pedicle. Many more of the cells have lost their polarity, and it is no uncommon thing to find two nuclei in one cell, one at the outer and the other at the inner end. Numerous regular mitotic figures are seen placed at the inner ends of the cells.

All these changes indicate a tendency on the part of the cells to become undifferentiated. The cells of the glands near the pedicle are obviously differentiated mucus-producing cells, and as such are fulfilling a definite physiological function. Many of the cells of the glands near the surface have seemingly ceased to perform the function of producing mucus, and are obviously more concerned with proliferation, becoming more and more undifferentiated.

Of the changes in the interglandular tissue with increase in size of an adenoma there is little to be said; the structure is essentially the same, but the gross amount increases *pari passu* with the growth of the glandular tissue.

**Pathogenesis and Evolution.**—The underlying cause leading to the formation of adenomata still remains uncertain, and no specific work has been done on the problem up to the present time. Two hypotheses come under consideration—first, the inflammatory, and, secondly, that concerned with primary epithelial change. In support of the former is the strong evidence of the development of adenomata in chronic inflammatory conditions, e.g., multiple polyposis, the demonstration of ova of the *Oxyuris vermicularis* in the bases of adenomata, the presence of small round-celled infiltrations and other types of cells indicative of a chronic inflammatory process, and the development of the tumours mostly in those parts of the bowel which are most liable to irritation, i.e., sigmoid and rectum. On the other hand, Hauser and Bardenheuer believe that a primary change takes place in the epithelium of normal glands, and that the inflammatory reaction seen in the tumour is secondary in nature. The latter author states that the development of the tumours in places in the bowel most liable to irritation is only of significance in their further development. Against the first hypothesis, and supporting the contention of the secondary nature of the inflammatory process, is the fact that the mucous membrane in the neighbourhood of all the adenomata examined showed no sign of any inflammatory reaction, but was perfectly normal in appearance. This fact has also been observed by Quénu and Landel, Hauser and Bardenheuer.

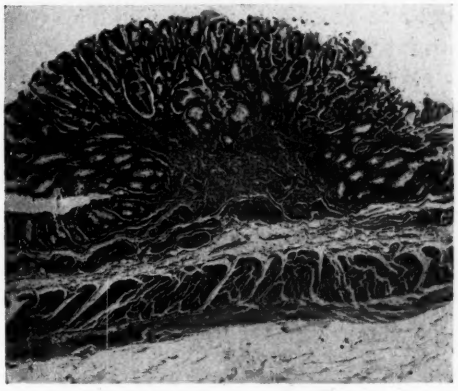

FIG. 80.—Early stage in development of a pedunculated adenoma. (× 15.)

It is comparatively easy to follow the development of an adenoma. At one point in the mucosa there commences a hypertrophy and hyperplasia of the glandular tissue, causing a slight thickening of the mucosa which forms a small fold. Then appears the first suggestion of a pedicle—a slight upward projection of the submucosa (*Fig.* 80). The larger-sized tumours simply

show an exaggeration of the preceding characters (*see Figs.* 77, 78). The glandular tissue has increased in amount, and rests upon a central stem or pedicle provided by the submucosa covered by mucous membrane continuous with that lining the bowel. The development of the pedicle is usually regarded as being due to two factors—first, the drag upon the tumour occasioned by the constant passage of fæces over it, and, secondly, the peristaltic contractions of the bowel endeavouring to expel what is virtually a foreign body.

The increase in the size of the tumour remains to be considered. Quénu and Landel " testify to a centre of proliferation " situated in the immediate neighbourhood of the pedicle, stating that new glands form in this area and migrate outwards towards the periphery, and that therefore the glands near the pedicle are youngest, while those near the surface are the oldest. The writer, however, holds the opposite opinion. He believes that once the glands have reached the stage of proliferation the tumour increases in size by the formation of new glands, so that the youngest glands are near the periphery. This opinion is held because of the general appearance of these glands—the cells are less differentiated, and therefore younger than those of the glands near the pedicle, while the glands themselves are often seen to be segmenting and thus younger ones are being continually formed.

**Adenomatous Polypi and their Relation to Malignancy.**—The fact that with the growth of the adenoma the cells tend to become less differentiated has been mentioned. Can this process become so pronounced that the cells may be called malignant, i.e., cells subserving no useful physiological function, but primarily concerned with the embryonic function of proliferation ? This is a most important consideration from the standpoint of surgical treatment of adenomatous polypi, and it is a question regarding which histological evidence of its affirmative answer will be submitted.

Seeing that the further from the pedicle the more advanced are the cellular changes, it is in the glands at the periphery of the adenoma that one must search for malignant (carcinomatous) change if it is to be found. The cases of adenomata, particularly the multiple variety, associated with carcinoma which have been recorded in the literature are so numerous that it has become the general opinion among pathologists and surgeons that the adenomata are definitely capable of undergoing malignant change. This conclusion, however, appears to rest mainly on clinical rather than histological grounds, the association of the two conditions being taken as the complete proof. Bardenheuer, and, later, Quénu and Landel, were the first authors to bring forward histological evidence of the malignant transformation of adenomatous cells. They showed that scattered throughout the larger adenomata were areas definitely carcinomatous in character. In this connection Lockhart-Mummery says : " The most important factor in connection with simple adenomata, whether of the single or multiple variety, is that they show a marked tendency sooner or later to become malignant, i.e., for the cells to penetrate the basement membrane and invade the surrounding structures. I am of opinion that all adenomata of the rectum eventually take on malignant change, and in the great majority of cases in which large adenomata have been removed, malignant change has been found to have already occurred in some part or other of the tumour. So marked is this tendency

for simple adenomata to become malignant, that personally I look upon adenomata as merely a stage in the development of malignant disease, and regard simple adenomata of the rectum as a definitely precancerous condition. It follows from this that on no account should a single adenoma of the rectum be allowed to remain, even if it is causing no troublesome symptoms; it should be freely removed as soon as possible ".

FIG. 81.—Large pedunculated adenoma of sigmoid colon.

Tuttle believes that the chances are that in about three out of four cases of multiple adenomata malignancy occurs in some of the growths sooner or later.

Of the cases examined in this series, in none of the adenomata of the multiple variety was carcinomatous change found—possibly because they had not attained a sufficient size in their development. The change was met with in three adenomata; all were single, all were about 4 cm. in diameter, and all were situated in the sigmoid colon; two of them were attached to the wall of the bowel by large, long pedicles (*Fig.* 81), while the third was sessile.

FIG. 82.—Carcinomatous area at periphery of adenoma shown in *Fig.* 81. (× 13.)

*Fig.* 82 is a photomicrograph of a section taken from the periphery of one of the large pedunculated adenomata mentioned above. In it are seen glands which as regards irregularity of size and shape, granular appearance of the cells, etc., conform to the description given of many of the glands which are to be seen towards the periphery of any fairly large adenoma. In these, though the cells have been undergoing a marked degree of proliferation, there is yet nothing to indicate a malignant change. There is, however, one area which obviously possesses characteristics different from the remainder of the section. Acini are seen in which are numerous cellular ingrowths; these

unite with others and so form epithelial partitions dividing up the acini irregularly. The cells are devoid of differentiation, and it seems as though the acini contained irregular masses of multinucleated protoplasm. The nuclei have proliferated and are arranged throughout the granular protoplasm in a most disorderly manner. They are irregular in shape and staining reactions, and several irregular mitotic figures are seen. Regular mitosis is seen occasionally in normal glands of mucous membrane. It is characteristic of epithelium which, for some reason—e.g., in benign tumour formation or in response to inflammation—is undergoing proliferation. Irregular mitosis, on the other hand, means that proliferation is so rapid that it is only seen in cells which are concerned with the embryological function of propagation; these of course being considered malignant. The nuclei are also distinctly larger than those found in the other acini; they stain more darkly, and are richer in chromatin bodies. (*Figs.* 83, 84.)

FIG. 83.—High magnification of edge of carcinomatous area, showing contrast between its cells and those of the adjacent benign alveolus. Absence of basement membrane and invasion of the connective tissue by malignant cells. (× 150.)

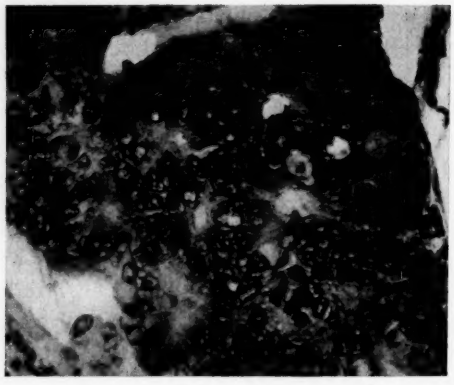

FIG. 84.—Higher magnification of carcinomatous area, showing irregularity in size and undifferentiated nature of the cells and mitotic figures. (× 240.)

At one part the basement membrane has completely disappeared and the cells are invading the surrounding connective tissue. Opinions differ as to the significance of the disappearance of the basement membrane. Lockhart-Mummery, Cornil, Ranvier, and David consider it to be pathognomonic of malignancy; Quénu and Landel state that they have only found the basement membrane in benign adenomata in a very inconstant manner, so that they do not attach much importance to its presence—or absence; Broders also holds the latter view.

Several sections taken from the periphery of this adenoma showed in nearly every case one or more small areas of carcinomatous change, while the glands and cells of those cut near the pedicle were all essentially benign. In the second pedunculated adenoma, similar in size to the first, malignant areas differing but slightly from that described above were discovered in sections taken from the periphery.

The third adenoma in this series in which malignancy was discovered was sessile, but similar in size to the others. Macroscopically one would have described it as a carcinoma, and it was not until it was examined microscopically that it was found to be an adenoma which had undergone considerable malignant change. In the section (*Fig.* 85) are seen many acini lined by well differentiated mucus-producing cells. Side by side with these, at the periphery of the tumour, are acini which may be safely designated malignant—irregular in outline, with numerous epithelial vegetations projecting into their lumina, great multiplication of nuclei which are larger than those of the mucus-secreting cells and stain irregularly and more deeply. It is interesting that in the same acinus as those cells which are apparently malignant are others quite well differentiated and apparently benign.

In the base of the tumour many irregular acini were seen invading the muscular coat of the bowel. In some of these, the cells, although showing signs of proliferation, were yet well differentiated and mucus-producing, and in fact could only be called malignant because of the position they occupied, having obviously infiltrated the tissue to get there. Thus it would seem that malignant cells which can infiltrate and destroy tissue, though usually having been bereft of their normal physiological function, yet in some instances may retain it in greater or less degree, thus adding to the difficulty in some cases of deciding whether certain cells are, or are not, malignant. The basement membrane is present in some of the glands and not in it is sometimes seen in one part of a gland others, so that it is not possible to lay great stress upon its presence or absence. Again, and is absent in another. When it is absent, the cells can be seen definitely invading the surrounding connective tissue. Considering the mixture of innocent and malignant acini seen, I think one can safely say that the tumour was primarily an adenoma which had undergone malignant change.

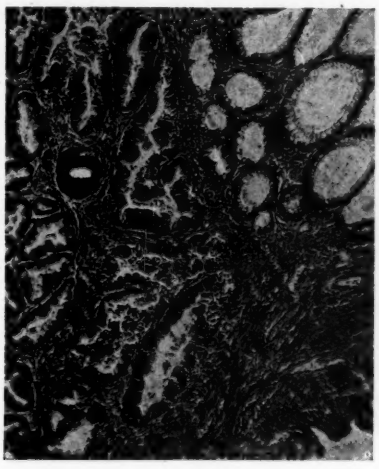

FIG. 85.—Benign and malignant glands in large sessile adenoma. (× 36.)

**The Association of Adenomata with Carcinomata.**—In the 36 cases of this series in which adenomatous polypi were found carcinomata occurred in 13. None of the adenomata in these 13 cases showed carcinomatous change. However, it must be stated that none of them had grown to anything like the large size of those mentioned in which carcinomatous change was found.

Considering the large number of carcinomata of the large bowel encountered without adenomatous polypi being present, the association of the two conditions in the same intestine is not very frequent, and it would seem that although carcinomata occur which are undoubtedly the outcome of malignant change taking place in adenomata, yet the majority arise *per se*. An interesting specimen bearing on this subject was found in this series. In the

wall of the sigmoid colon there was a flat hard plaque 2·5 cm. in diameter, more or less circular in outline and raised about 2 mm. above the surface of the mucosa. *Fig.* 86 illustrates the appearance of a section taken from the edge of the tumour. It is obviously a carcinoma. The abrupt change between the normal mucous membrane and the carcinomatous acini is well shown. The acini have grown downwards and invaded the muscular coat of the bowel. One striking thing about the section is that there is no suggestion of simple adenomatous formation, the acini without exception being carcinomatous in character. This tumour the writer believes to be a small primary carcinoma.

FIG. 86.—Primary carcinoma of intestine. (× 15.)

The following tables show the relation between number and sex and the age incidence of the cases of polypi of the large intestine in this series.

*Table VII.*—NUMBER AND SEX INCIDENCE.

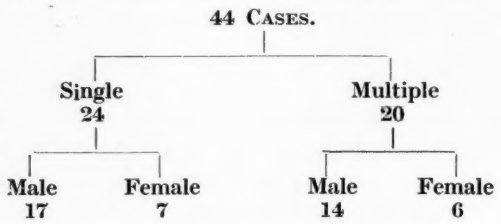


*Table VIII.*—AGE AND SEX INCIDENCE.

| AGE | MALE | | FEMALE | |
|---|---|---|---|---|
| Years | Single | Multiple | Single | Multiple |
| 20–29 | 0 | 2 | 0 | 0 |
| 30–39 | 1 | 0 | 2 | 3 |
| 40–49 | 3 | 0 | 2 | 1 |
| 50–59 | 5 | 2 | 2 | 0 |
| 60–69 | 6 | 6 | 1 | 1 |
| 70–79 | 2 | 4 | 0 | 0 |
| 80–89 | 0 | 0 | 0 | 1 |

In this series, therefore, single polypi were met with more frequently than those which are multiple. From *Table VIII* it is apparent that polypi may be found in the large intestine at almost any age. They were in greatest number between the ages of 60 and 69.

## CONCLUSIONS.

1. Polypi occurred with much greater frequency in the large than in the small intestine, the proportion being 4 to 1 approximately.

2. In the small intestine polypi due to glandular hyperplasia were the commonest type.

3. In the small intestine the ileum, and in the large intestine the sigmoid, were the most frequent sites of occurrence of polypi.

4. In both small and large intestines polypi occurred more frequently in males than females.

5. In the large intestine the adenoma was the type of polypus most frequently met with.

6. Adenomatous polypi were found capable of undergoing malignant change.

7. Malignant change was demonstrated to commence at the periphery of the tumour.

8. This change was not observed in any adenoma which was smaller in size than a walnut.

## APPENDIX.

With a view to gaining an idea of the appearance of a polypus when magnified a large number of times, reconstruction in wax of one of them (a pedunculated adenoma) was undertaken. Below is a description of the apparatus and technique used.

The wax was a mixture of three constituents, viz., beeswax, paraffin wax, and resin, in the proportion of 3 : 2 : 1. The aim is to produce a wax which will not split when cut by a knife, and for this reason beeswax and paraffin wax alone or together are unsatisfactory. The mixture is heated until it melts. Plates of this material of the desired thickness are made and allowed to harden by cooling.

The apparatus used consists essentially of two parts, an iron roller and a metal plate, which must be horizontal. The roller is a cylinder 18 in. long and $3\frac{1}{2}$ in. diameter, having at each end a cylindrical projection of $1\frac{1}{2}$ in. diameter used as a handle. The roller rests on a stand placed just behind the horizontal plate, over which it can be easily pulled forwards, and under it is a multiflame Bunsen burner. The horizontal plate measures 16 in. by 14 in., and is fixed between two vertical metal sides about $\frac{1}{4}$ in. in thickness. Each of these has two vernier gauges, one near either end, and by means of these the plates are raised above the level of the horizontal plate, the exact amount corresponding to the required thickness of the wax plate.

The roller and horizontal plate are now heated, the former by its special burner, the latter by pouring on alcohol and setting it alight. After the alcohol has burned, the residual water is wiped off and the plate smeared well with xylol, toluol, or oil of turpentine. A sheet of waxed paper of width equal to that of the plate is then placed on the latter and smoothed out with xylol. The melted wax is next poured on to the plate, the roller brought forward from its stand and moved forwards and backwards on the two vertical plates, flattening out the wax in a uniform manner, and then replaced on its stand. As soon as the wax has solidified, it is cut with a knife along its whole length just internal to the vertical plates. The waxed paper is raised in front along its whole breadth by means of a knife; the raised part is then seized, the sheet of wax being lifted off the plate (this being done with ease owing to the previous use of xylol) and set aside to harden. When it has done so, the waxed paper is stripped off it, this operation being performed with little difficulty, owing to the smearing of the waxed paper with xylol.

To obtain the optimum temperatures of the melted wax, the roller, and the horizontal plate is a matter of experiment. If they are too low, the wax will harden before one has time to use the roller; if too high, an enormous amount of time will be wasted waiting for the wax to solidify before taking it off the metal plate.

The polypus was taken, fixed and embedded in the usual manner, and serial sections were cut and stained. Each section was 10 micra in thickness and the magnification 30. Then in order to utilize every section a wax plate 0·3 mm., i.e., 30 × 0·01 mm., in thickness would be necessary. However, it was found that the smallest satisfactory thickness of wax with which to work was 1·5 mm., and so only one in every five of the serial sections made could be used, as otherwise the model would be five times too long in proportion to its width.

A microscope was fixed in a horizontal position, and the ordinary eyepiece replaced by one with a right-angled prism so that the horizontal beams of light were reflected downwards in a vertical direction on to a sheet of paper. A slide was inserted, and, after the image of the polypus had been focused on the paper, its outline was drawn. The paper was laid on one of the wax plates, and the outline

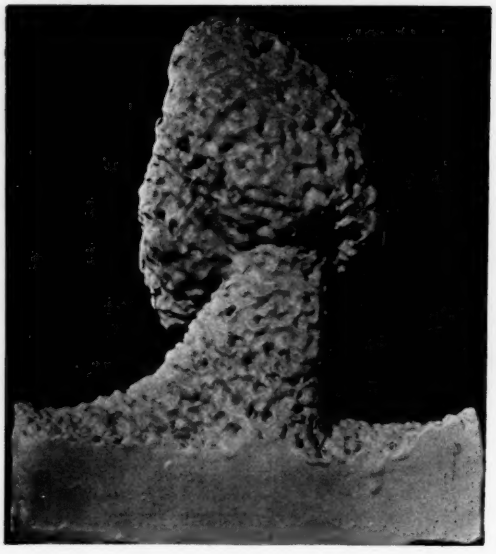

FIG. 87.—Model of pedunculated adenoma.

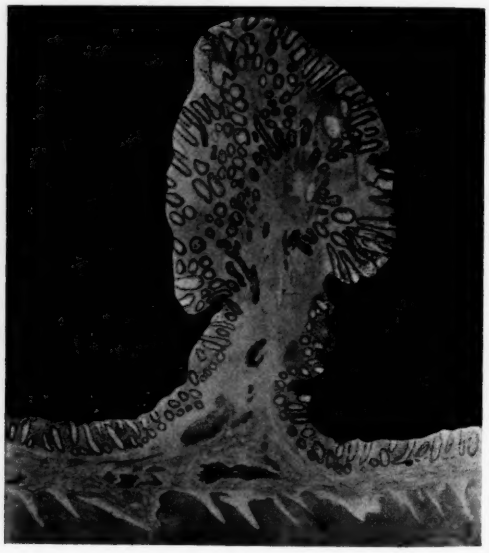

FIG. 88.—Model showing same adenoma as in *Fig.* 87 on section.

of the polypus traced with enough pressure to make it appear on the plate. After the removal of the paper the wax was cut with a tenotome along the outline of the polypus.

To ensure accuracy in the assembling of the wax plates which had been cut out, three points were taken as far apart as possible on the first outline drawn. Before the second sheet was traced on the wax it was held up to the light under the first sheet, the two outlines being easily seen. The latter was correctly superimposed on the former, and the three points were traced through. This process was carried on throughout. After each outline had been traced on the wax plate, the three points were stamped through, and afterwards holes were made through the wax by a sharp pencil at these points. A thread was passed through each corresponding hole throughout the whole thickness of the model, and thus correct superimposition of each wax plate on the preceding one was ensured.

After the plates were assembled they were pressed together and the remaining lengths of thread cut. The whole surface was then treated with an electric cautery,

making the plates adhere to one another and smoothing out the irregularities of the model.

The completed model was then painted in as natural colours as possible (*Fig.* 87). The depressions represent the mouths of the glands which open on the surface, and those of the tumour are seen to be larger than those of the mucous membrane covering the pedicle and lining the bowel. *Fig.* 88 is a photograph of a second model made to show the same polypus on section.

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# SOME GENERAL DISEASES OF THE SKELETON.

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(*Being the Presidential Address to the British Orthopædic Association, Oct.* 22, 1926.)

THE group of affections which it is proposed to consider to-day has a peculiar fascination, since little or nothing is known as to the causation of many of them, and the diagnosis and treatment of the individual cases are made an occasion of most interesting consultations between the physician and the surgeon. The light-heartedness inspired at the start by the interest of the subject chosen for this address soon vanished when an intensive study was commenced of the pile of radiograms and case records that had been collected with the help of many generous colleagues. The further the study proceeded, the more obvious became the size and complexity of one's task. At present the investigation of the material available has done little more than reveal some of the difficulties with which the subject is surrounded.

Many of the doubtful questions will not be solved for years to come; not indeed until much work has been done by the biochemist, as well as the pathologist and clinician. All we can manage to do to-day is to refer to a few of the problems.

We are gradually learning more about the normal process of ossification and the growth of the bones. Calcium and phosphorus in proper amounts are, of course, essential to this process. R. Robison[1] has worked at the chemistry of the subject, and has shown that an ester—hexose monophosphoric ester—plays an all-important part in the conveyance by the blood of the calcium, with which it forms soluble salts. This combination of the ester and calcium is hydrolysed by a ferment present in ossifying cartilage, calcium phosphate with some calcium carbonate being deposited around the osteoblasts. Now this ferment is only found, except in minute quantities, in two other places in the body besides ossifying cartilage, viz., the intestines and the kidneys, where, it has been suggested, it plays a part in the absorption and excretion, respectively, of calcium. It is noteworthy that the ferment is absent from muscles. This piece of work opens up great possibilities for research and sets one thinking. Is it possible that the presence of this ferment in abnormal situations, or in abnormal quantities in normal situations, is responsible for some of the affections we are now considering? What bearing has this research work on delayed union of fractures about which we are so anxious for further information? Has it anything to do with the question of non-union in an apparently healthy person where no anatomical explanation can be found? Is its action inhibited or destroyed by synovial fluid? To go further afield, is the presence of the ferment in abnormal situations responsible for the calcification in joint capsules, muscles, and tendons that sometimes follows trauma, and for the deposit of lime salts in the walls

of blood-vessels and other tissue? Further reference will be made to this ferment later.

For the normal development of bone three things are necessary: (1) A proper cartilaginous or membranous scaffold or foundation for the bone; (2) The deposit of the necessary inorganic salts; and (3) A sufficient number of normal bone-forming cells. If the scaffold is not set up, no bone develops, and if extra scaffolding is erected, we get supernumerary bones or digits formed, or even complete limbs. Abnormality in the structure of the scaffold or in its adaptability to the changes involved in the formation of bone might be expected to play a part in the production of some of the gross errors of skeletal development that we meet with; such abnormality is regarded by Jansen[2] as the fundamental cause of some of the congenital conditions we are considering. Deficient deposit of lime salts in a developing bone may arise either from a scarcity of these salts in the circulating blood, or from some inability on the part of the individual to eliminate them. Much research work has been done on calcium metabolism, but much remains to be done. We know that in rickets the intake and excretion of calcium are increased and the blood calcium is usually normal in amount, though the phosphorus in the blood is often diminished. In osteomalacia of all kinds the excretion of calcium is said to be much increased without any increase in the intake, so that the amount in the circulating blood is diminished. Cod-liver oil, by the way, has no effect on the excretion of calcium. In osteogenesis imperfecta neither the intake nor the excretion is increased, while the blood calcium may be either above or below the normal 9 to 11 mgrm. per 100 c.c. In two ante-natal cases of this affection at the Hospital for Sick Children the figures for the blood calcium were 12·3 and 5·45 respectively. In renal rickets the blood calcium is said to be diminished. Interesting though these findings are, they do not help us much, at present at any rate, with the etiology of these diseases. With regard to the third factor, the osteoblasts, again we find a great field for research work. Lawford Knaggs[3] in cases of osteogenesis imperfecta found a marked deficiency in these bone-forming cells, the dearth of which he regards as the essential cause of all forms of this affection. Fat-soluble A, which plays such an important, though at present obscure, part in the normal growth and calcification of bones, is not as simple a vitamin as we used to think. Having changed into twins, it has now become triplets, the names of the brothers being A, D, and E.

The consideration of the large number of recognized general bone affections demands a classification of some kind, and I venture to suggest the following:—

*Table I.*—General Affections of the Skeleton.

*Congenital Developmental Errors*—

- Osteogenesis imperfecta
- Diaphysial aclasis
- Dyschondroplasia
- Achondroplasia
- Craniocleido dysostosis
- Ateleiosis
- Myositis ossificans idiopathica

*Errors of Metabolism*—

- Scurvy
- Rickets
- Renal dwarfism
- Dwarfism associated with:
  1. Cœliac disease
  2. Pancreatic disorder
  3. Hypertrophic cirrhosis of liver

*Endocrine Errors—*

- Gigantism
- Acromegaly
- Mollities ossium
- Osteomalacia
- Dwarfism and infantilism of various types (*see Table II*)

*Affections of Unknown Origin—*

- Fibrocystic disease
- Leontiasis
- Osteitis deformans
- Arachnodactyly

*Multiple Neoplasms.*

Though no doubt this classification is open to criticism on some points, and its revision will be necessary in the light of further research, it is hoped that it will serve as a foundation. We may now proceed to discuss a few points of interest in connection with some of the affections cited in this table.

## CONGENITAL DEVELOPMENTAL ERRORS.

**Osteogenesis Imperfecta.**—Thomson[4] divides these cases into two groups —namely, the intra-uterine disease which is frequently fatal, and that which

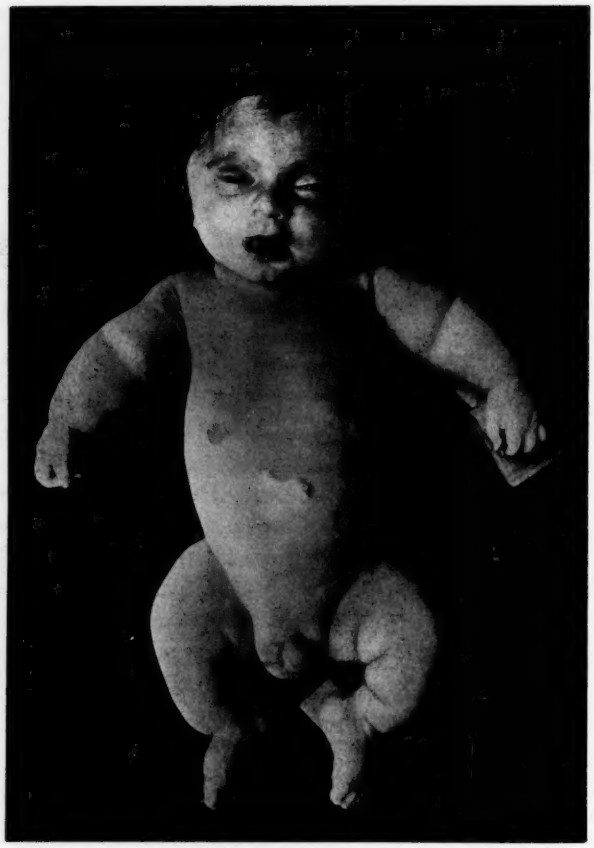

FIG. 89.—Osteogenesis imperfecta, Type 1, with short limbs. Age 2 days. (Under Dr. G. F. Still.)

develops later and is associated with blue sclerotics. There seems to be no sharp distinction between these two groups. A study of the histories and

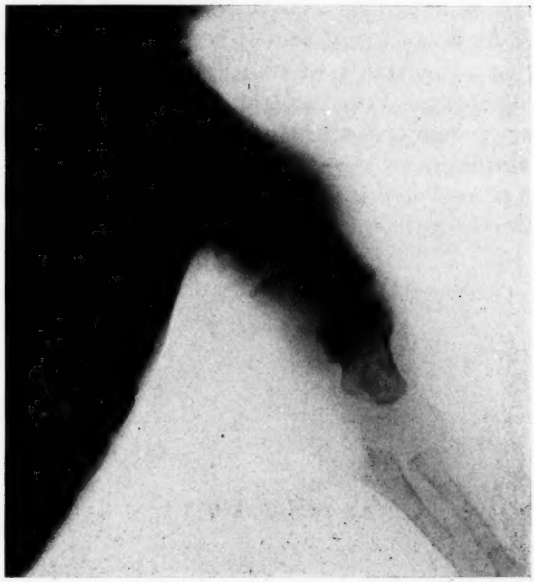

FIG. 90.—Osteogenesis imperfecta, Type 1, age 10 weeks. (Under Dr. Poynton.)

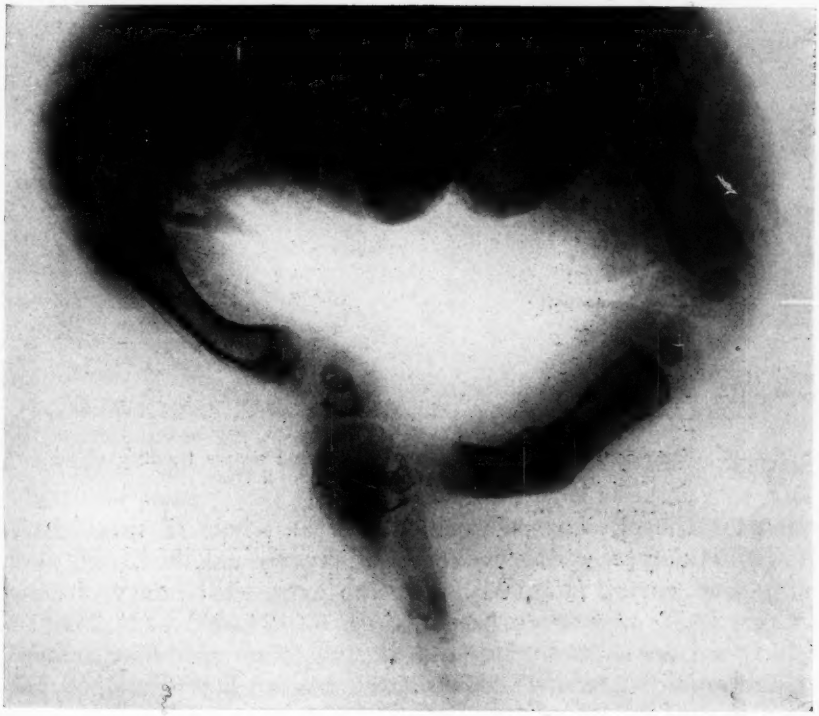

FIG. 91.—Same case as *Fig.* 90, showing legs.

radiograms of some 18 cases has shown that it is possible to recognize four clinical types, but it is not suggested for an instant that these represent four different diseases. As a matter of fact it seems more than likely that the first three at any rate are the result of one and the same disease. These types are: (1) The young infants born with multiple fractures—the so-called fœtal type—and showing a marked stunting of the limbs as compared with the trunk (*Fig.* 89). Radiograms show the femora and humeri, and less frequently the tibiæ, to be short and strikingly thick in proportion to their length. These bones show one or more fractures with abundant callus, but this hardly seems

FIG. 92.—Osteogenesis imperfecta, Type 2, showing simple fragility of bones. Girl, age 2½ years. (Under Mr. Tyrrell Gray.)

to account for the obvious enlargement of the whole of their shafts (*Figs.* 90, 91). (2) The type with slender bones, poorly calcified, and showing an abnormally thin cortex (*Fig.* 92). (3) The type with honeycombing of the bones, which bend as well as break (*Figs.* 93, 94, 95). (4) The type with bones which are denser than normal and have their medullary canals diminished or obliterated—the so-called 'marble bones', first described by Albers-Schönberg in 1904.[5]

Cases of the first type rarely survive for long. The second type includes

the great majority of the cases seen in childhood, and occasionally in adolescent and adult life. Some are born with a fracture or two; some develop the tendency to fractures after birth; in some the bone fragility seems to disappear after a time. Some of these cases go from bad to worse, develop marked deformities, become bedridden, and sooner or later succumb to intercurrent disease. The severe cases, with the characteristic broad skull which bulges laterally above the ears so that the latter face downwards and outwards, belong either to this group or to the third group with honeycombed bones. I am not prepared to say at present whether the third type is or is not a late development of the second. Many, but not all, of these severe cases have blue sclerotics, a feature which is not, by the way, confined to those with a post-natal onset. There seems to be no definite line to be drawn either between the first two groups or between the second and third. Softening and bending are seen in some cases and not in others, in addition to the fractures and the deformities resulting therefrom. As to the cause we are still in the dark. Lack of vitamin A during pregnancy, as suggested by Thursfield,[6] cannot, I think, be the cause, for, if this theory were correct, the disease should be more amenable to treatment. Light treatment does not affect these cases materially. Knaggs[7] attributes all the cases to the same cause, namely, a failure of the evolution of the highly specialized osteoblast, which he found markedly deficient in numbers, but he can suggest no reason for this failure. It would be of great interest to know whether there is any lack of Robison's ferment in the ossifying cartilage in these cases.

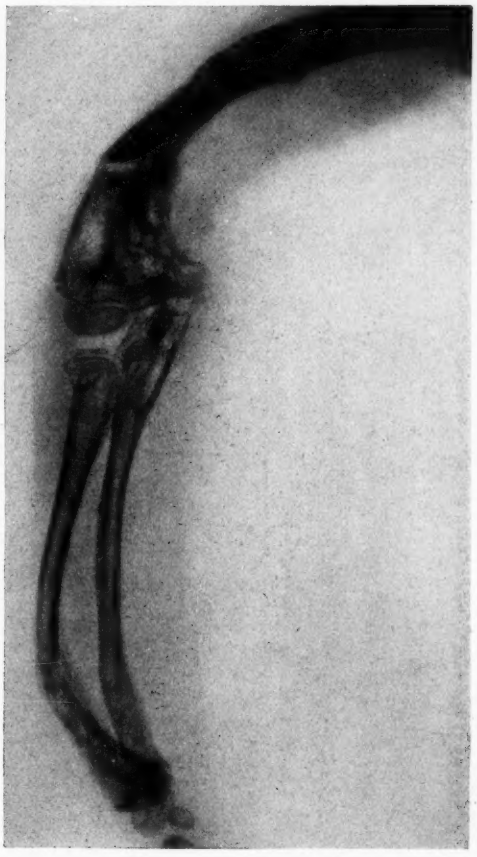

FIG. 93.—Osteogenesis imperfecta, Type 3, with honeycombed bones. Girl, age 5½ years. Upper limb.

'Marble bones' are in quite a different category, the only resemblance they have to the three preceding groups being the fragility of the bones (*Figs.* 96, 97). The feature of the group is a great excess of lime salts in the bones. It appears to be a rare disease, which may be met with in both sexes and at all ages—the ages of the recorded cases ranging from 3 weeks to 43 years. In addition to the bone changes, calcareous deposits are seen in the dura and in the ligaments. The diagnosis is revealed by the X rays, which show greatly increased density of all the bones, with opaque patches of calcareous

material, thickening of the cortex of the long bones, and reduction of the medullary canal. I have only one case which seems to be of this type, though it does not show the more striking radiographic appearances of the well-marked disease. A girl of 12 sustained her first fracture of the tibia when 5 years of age, and in the succeeding five years one or other tibia was fractured seven times. All her bones are abnormally dense and structureless, the cortex of the shafts of the long bones is unduly thick, and the medullary

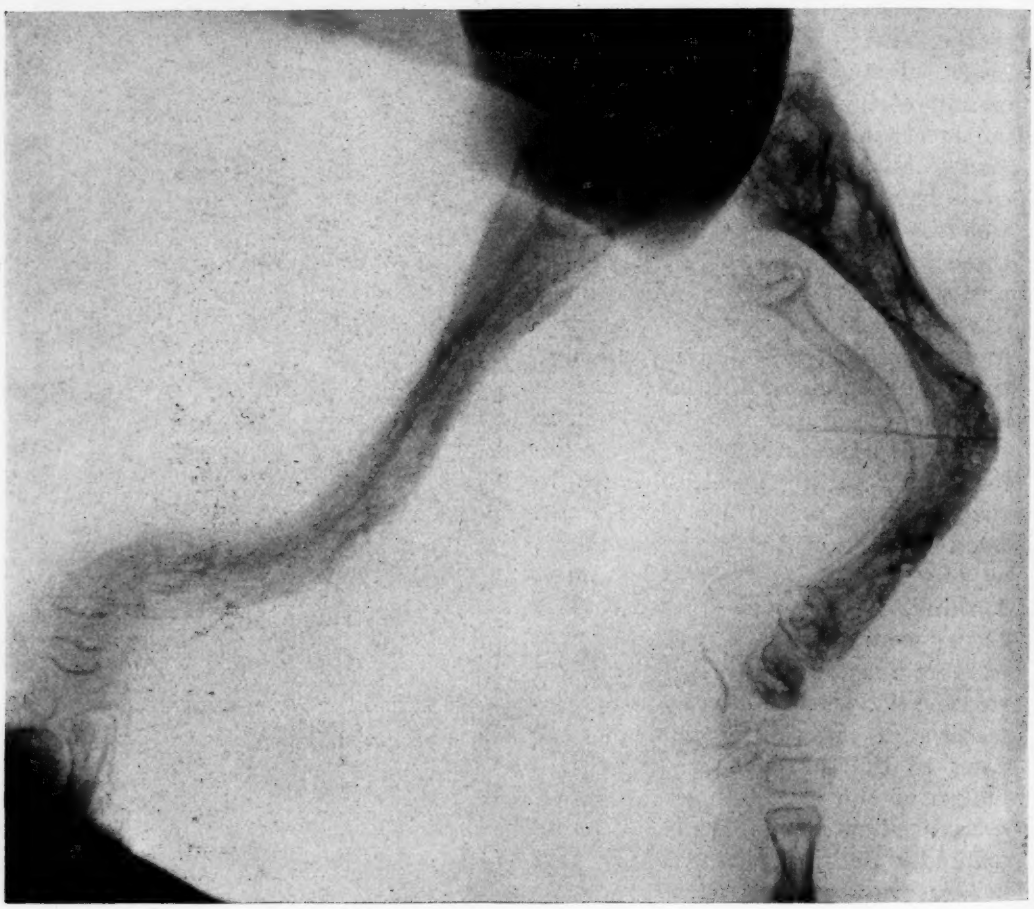

FIG. 94.—Same case as *Fig.* 93, showing legs.

canals are reduced in size. The tibiæ gave way at the same spots on each occasion. Union took place slowly and was obviously imperfect. The head, though not typical, was broad, and the chin was pointed. The sclerotics were definitely blue. Is it possible that an unusual supply of the ferment discovered by Robison is responsible for this excessive calcification? This would mean the abnormal presence of this ferment in the bone already formed, as well as an excess in the ossifying cartilage, for the abnormal density of the bones persists in some cases even in adult life. Deficiency in the number

or nature of the osteoclasts present is another possible explanation, I suppose.

**Dyschondroplasia.**—This name is usually applied to a condition described by Ollier[8] in 1898 and characterized by dwarfing of the affected limbs, coupled with irregular ossification at the ends of the diaphyses of the long bones. It is commonly unilateral, but by no means always so. The ends of the affected

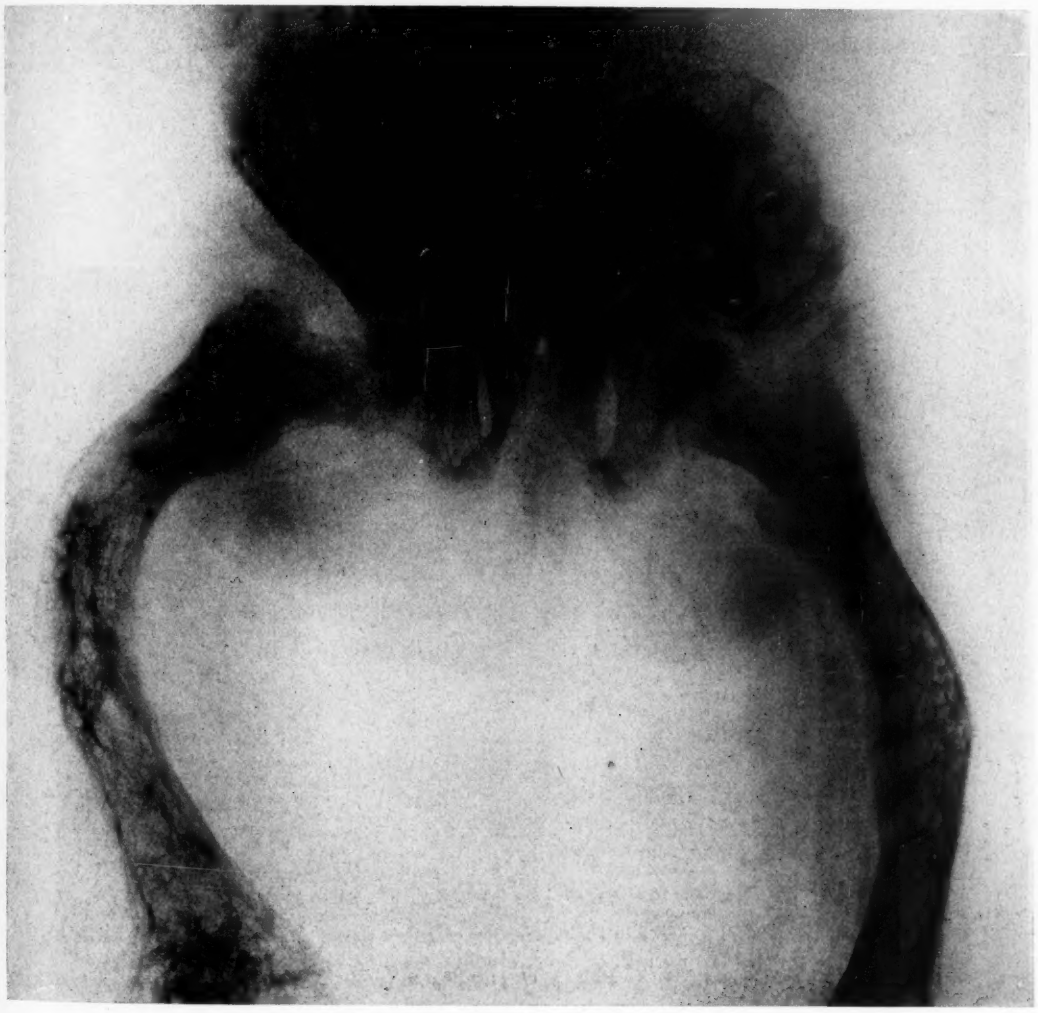

FIG. 95.—Same case as *Fig.* 93, showing pelvis and femora.

diaphyses are enlarged, and show a number of translucent areas which are filled with cartilage. The hands and feet are often affected, but the carpus and tarsus are normal. The disease is much more common in males, and is often hereditary. Although the contrary is said to be the case, I believe the affection is essentially congenital, though there may be comparatively little sign of it at birth. By the courtesy of Dr. Gordon Pugh I am able to give

radiograms of a case under his care (*Figs.* 98, 99). In this case, a boy of 12, the disease is practically confined to one side of the body. I would call particular attention to the combination of clear areas with opaque spots at the ends of the diaphyses in this case. The epiphyses appear to be fairly normal, and in this the case agrees with the usual description of the disease. In another typical case, however, we see, at the age of 3 years, mottling and honeycombing of the epiphyses associated with the usual changes at the ends

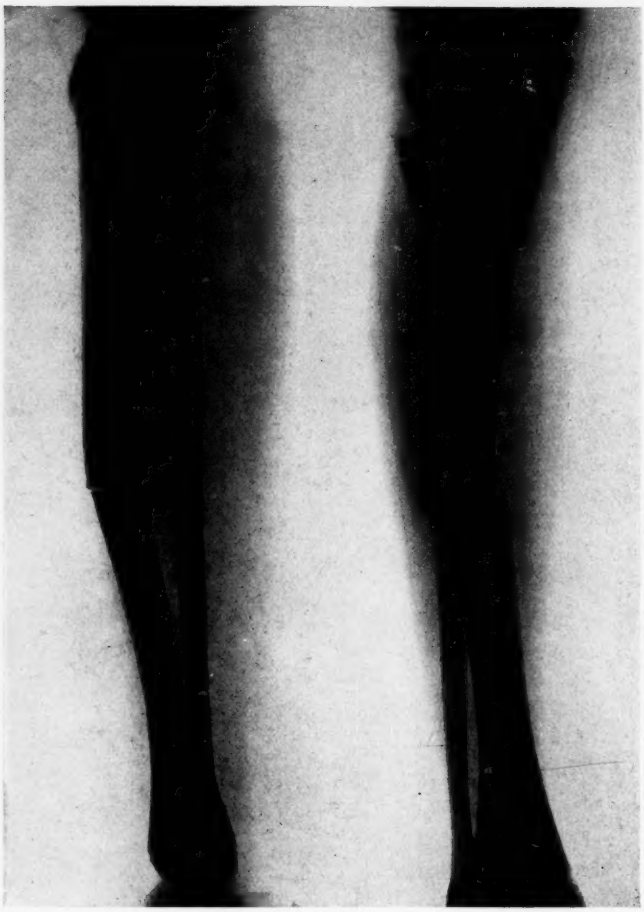

FIG. 96.—Osteogenesis imperfecta, Type 4, 'marble bones'. Girl, age 12 years. Tibiæ.

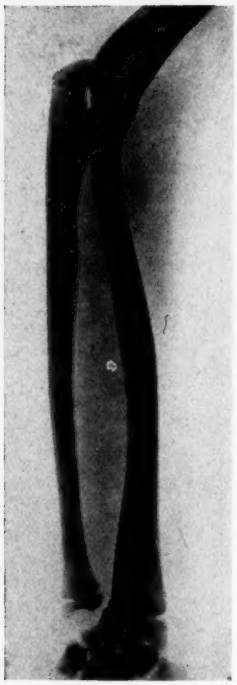

FIG. 97.—Same case as *Fig.* 96, showing forearm.

of the diaphyses (*Fig.* 100). In 1924 Voorhoeve,[9] of Amsterdam, published three cases, members of one family, with curious striation of the ilia and the diaphyses of the long bones somewhat similar to that seen in a case published by myself last year.[10] He analyses the literature on the subject most carefully, and points out how frequently striation, clear spots, and spots of increased density can be made out in the radiograms of a single case, though one or other of these features may predominate. He argues that all these

changes are due to errors in the ossification of cartilage, and that therefore they should all be placed in one class, the dyschondroplasias. He even includes the condition called by Albers-Schönberg 'osteopathia condensans disseminata'.[11] In this the dense spots, which may be few or many, are more regular in outline than those seen in a typical Ollier's disease, and are usually oval, with the long diameter parallel with the long axis of the bone. These spots have been seen in the epiphyses as well as the metaphyses, and, indeed,

FIG. 98.—Dyschondroplasia (Ollier's disease). Boy, age 12. Left knee showing clear areas, dark spots, and striation. (Dr. Pugh's case.)

FIG. 99.—Same case as *Fig.* 98. Left forearm.

in every bone except the ribs and the skull. These changes are always found by accident and are not associated with any symptoms. Though sceptical at first, I must confess that Voorhoeve seems to me to make out a fairly good case for his views. Even if we follow him and group together all these errors in the transformation of cartilage into bone under the title of dyschondroplasia, we must recognize several distinct types.

In this connection I should like to mention two cases showing mottling

confined to the short bones and to the epiphyses of the long bones. The first was an infant of one month with a congenital shortness of one leg (*Fig.* 101). The epiphyses of the long bones of the leg, and the tarsal bones, are represented by a number of discrete dense spots giving a curious stippled appearance. Unfortunately the case was lost sight of after its first visit to the hospital, but inquiries elicited the fact that it died when nine months old. The other case was under the care of Dr. Eric Pritchard at the Infants' Hospital, who kindly allows me to refer to it (*Figs.* 102, 103). The child, age 2 years, was

FIG. 100.—Dyschondroplasia (Ollier's disease). Boy, age 3. Note changes in epiphyses as well as shafts.

FIG. 101.—Congenital short leg. Child 1 month old, showing stippled epiphyses and tarsal bones.

decidedly small for its age, and showed some of the stigmata of rickets. The limbs were not disproportionately short, but there was contracture of both elbows and knees. The X rays showed the epiphyses to be stippled, much as in the preceding case, and the shafts of the long bones to be stouter than normal, with trumpeted extremities. In this case the changes were bilateral, while in the other apparently unilateral. In both the ossification of some epiphyses and of some of the small bones had commenced much earlier than

FIG. 102.—Elbow of child, age 2 years, showing stippled epiphyses. ? Dyschondroplasia. (Under Dr. Eric Pritchard.)

FIG. 103.—Legs of same case as *Fig.* 102.

usual. It is difficult to fit these into the dyschondroplasia group, and it would seem necessary at present to regard them as examples of an entirely separate and, perhaps, a new affection.

Before leaving this group I must refer to the work of Bentzon,[12] who regards Ollier's disease as the reaction of the bones against certain disorders of the innervation of their blood-vessels. He called attention to the fact that the clear areas in the diaphyses were arranged in fan shape, radiating

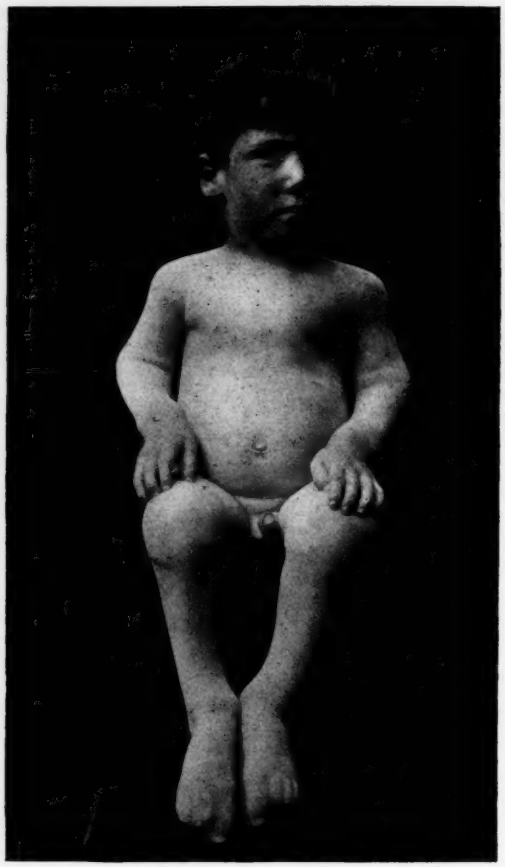

FIG. 104.—Boy, age 4½ years, showing enlarged epiphyses and other deformities of unknown origin. (Under Dr. Hugh Thursfield.)

FIG. 105.—Upper limb of case shown in *Fig.* 104.

from the point of entry of the nutrient vessel towards the epiphyseal line, i.e., along the lines followed by the vessels. In a series of 23 experiments in which he injected alcohol round the nutrient artery of the tibia in rabbits, he produced changes like those in Ollier's disease in 2. Shortening occurred, and clear areas appeared, and these he proved to be free from vessels and to be filled with cartilage. In another series of 30 experiments he destroyed the sympathetic with alcohol, but only in one did he obtain any result, a phalanx showing changes a month later.

**Achondroplasia and Craniocleido Dysostosis.**—One of the chief points of interest in connection with these conditions is the most ingenious and admittedly attractive theory of Murk Jansen.[13] This theory presupposes the existence of abnormal amniotic pressure during a short period, a mere matter of days apparently, in early fœtal life. This temporary pressure is said to produce permanent injurious effects on the portions of the skeleton which happen to be undergoing particularly active changes during the period of its operation. There seem to me great difficulties in the way of accepting this theory. We have no proof at present of the existence of this excessive amniotic pressure. Why should a pressure sufficient to produce such harmful

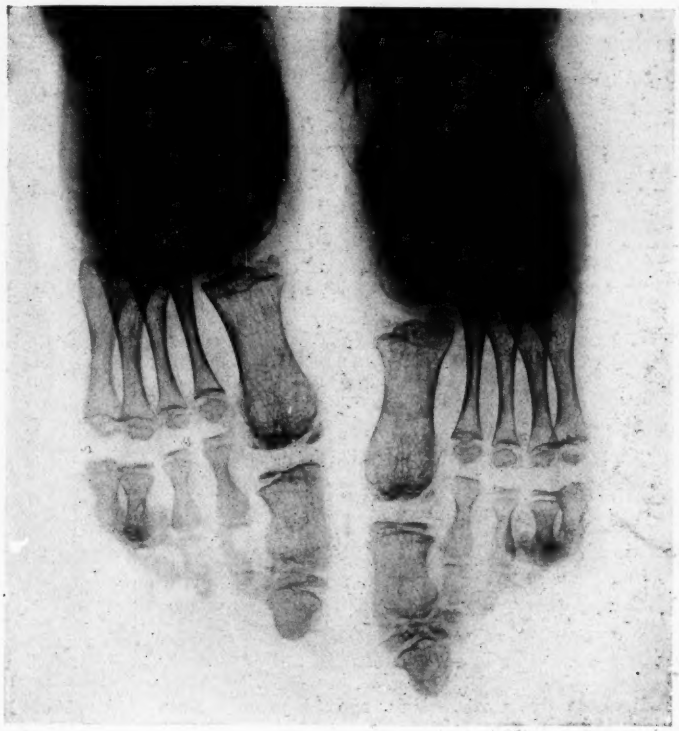

FIG. 106.—Feet of same case as *Fig.* 104, showing abnormalities of big toes.

effects exist only for so brief a period? Why should a temporary pressure produce such lasting effects?—for the error of development, whatever its cause, continues throughout the whole of the growing period of the subject affected. If a chance increase of pressure be the cause of achondroplasia, how is it that these cases are so singularly true to type? The epiphyses in achondroplasics are at least normal in size and well formed. The appearance of their centres of ossification may be either unduly advanced or unduly delayed. Why should the ossification at the epiphyseal line be so markedly affected while in other respects the epiphysis is well formed and apparently performing its functions?

Let us look at some radiograms of these conditions. In *achondroplasia*,

if one disregards the shortness of the diaphyses and the trumpeting of their ends, the most marked and really characteristic feature seems to me to be the position of the ossific nucleus in the epiphysis. This centre of ossification always appears close up to the end of the shaft of the bone, instead of in the usual situation, which is much nearer the centre of the epiphysis. The end of the shaft is usually funnel-shaped, and the epiphyseal centre is fitted into the hollow thus formed. It is rather doubtful whether, clinically, the epiphyses are really larger than the normal, or only apparently so. Comparing the knees of a case with those of a normal boy of the same age, it is obvious that the epiphyses of the achondroplasic are at least as large as the normal. The diminution in size of the big toe, emphasized by Jansen, appears to be due to shortness of the first metatarsal, a point

FIG. 107.—Craniocleido dysostosis. Boy, age 3½ years. Note absence of ossification of pubis. (Under Mr. Barrington-Ward.)

FIG. 108.—Forearm of same case as *Fig.* 107.

which may be contrasted with the diminutive first phalanx seen in myositis ossificans. Thomson[14] refers to two types with short limbs that are not achondroplasics. The first is congenital, and has a normal head, very convoluted auricles, and typical hands, the fingers being contracted and bent towards, not away from, the median line of the limb. The second type, described by Herringham and Drysdale,[15] is an hereditary condition acquired after birth. The hands become stunted. and the fingers thick, fleshy, and conical, but quite parallel. The phalanges are thickened, and the ossification of these and the carpus is much delayed. A case seen with Dr. Thursfield[16] is worthy of mention, since at the first glance it was not unlike an achondroplasic (*Figs.* 104–106). The striking peculiarities are: (1) Enormous enlargement of the epiphyses; (2) Enlargement of the big toes and the first

metatarsals, with limited motion in the metatarsophalangeal joint and ankylosis of the terminal joint; (3) Stiffness of the fingers; (4) Kyphosis and scoliosis; and (5) Coxa vara.

In *craniocleido dysostosis*, apart from the well-known abnormalities, we find others which have not, I think, attracted particular attention, and which seem to be very difficult to explain on Jansen's theory. In the case, age $3\frac{1}{2}$, which my colleague Mr. Barrington-Ward showed to this association two years ago, he called attention, among other points, to the complete absence of ossification in the pubis and in the carpal bones, and to the presence of epiphyses at both ends of several metacarpals and phalanges (*Figs.* 107, 108). In a case, age 6, which I showed before the association,

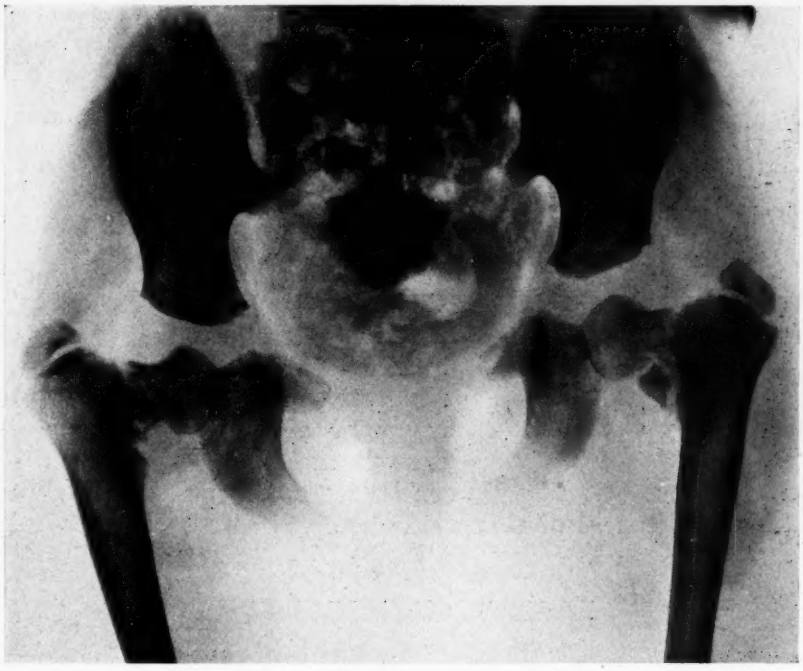

FIG. 109.—Craniocleido dysostosis. Girl, age 6. Note absence of ossification of pubis, and coxa vara of infantile type.

these abnormalities in the pubis and metacarpals are also seen (*Figs.* 109, 110), while in addition there is bilateral coxa vara of the cervical or infantile type. Mr. S. T. Irwin, who drew my attention to the true nature of this second case, kindly sent me radiograms of a case of his own where the pubis was unossified and there was bilateral coxa vara. In both these cases with coxa vara the heads of the femora encroach on the necks above in a quite abnormal manner, a point I have noted in many cases of infantile coxa vara unaccompanied by signs of dysostosis elsewhere. I find it very difficult to explain these abnormalities by any pressure theory. Entire absence of the pubis one could understand, but the pubis is not absent: it is present, but unossified. The condition of this bone in adolescent and adult cases of this affection I am not

at present able to state. It seems to me that the suggestion that achondroplasia is a reversion to an ancient pigmy type is probably correct, and dysostosis is a developmental error resulting from an influence of a similar kind.

FIG. 110.—Hand of same case as *Fig.* 109, showing abnormal ossification.

**Ateleiosis,** the cause of which is unknown, is the name given by Gilford[17] to the type of retarded growth which provides the majority of the professional dwarfs.

**Myositis Ossificans Idiopathica.**—This is unquestionably a congenital affection. In a considerable number of cases of this rare condition, partial or complete suppression of the proximal phalanges of the thumb and big toe has been reported, while other errors of ossification have been met with. These are unquestionably congenital. Though no case, I believe, has ever been recognized at birth, Sir Archibald Garrod met with it at 5 months,[18] and it usually becomes obvious in childhood. While in common with the traumatic variety the masses of bone later become attached to the normal skeleton, it displays this marked difference from the traumatic affection, that a mass of bone once formed never disappears. It would seem therefore that this affection can be regarded as a skeletal abnormality, the gradual development of which is determined before birth.

## ERRORS OF METABOLISM.

**Scurvy.**—Dr. Bertram Shires has pointed out to me, as a characteristic feature of radiograms in infantile scurvy, the presence of a dark line at the end of the diaphysis with a light band behind it, in addition to the subperiosteal shadows which may or may not be present. That scurvy is sometimes met with, even in these enlightened days, in older patients, was proved by the case, age 18, which Dr. Cassidy[19] showed before the Royal Society of Medicine three years ago. The most noticeable features in the radiograms were triangular patches of great density occupying a central position towards the ends of the diaphyses of the long bones. In a case of Dr. Pugh's[20] a similar diagnosis of scorbutic infantilism was suggested when it was shown in the Orthopædic Section of the Royal Society of Medicine.

**Renal Dwarfism.**—Chronic nephritis may be associated with, if not the cause of, several conditions of interest to us. First, dwarfism and infantilism. To these may be added changes at the epiphyseal lines similar to those of rickets. As the skiagrams show, these rickety changes may respond

to appropriate treatment as they do in ordinary rickets (*Figs.* 111, 112). I have known such improvement to be followed by a relapse which again yielded to sunlight and other treatment. Lastly—and I think we only see these changes in cases going downhill fairly rapidly, and when the bones are decidedly atrophic—abrupt deformities occur as a result of fractures, partial

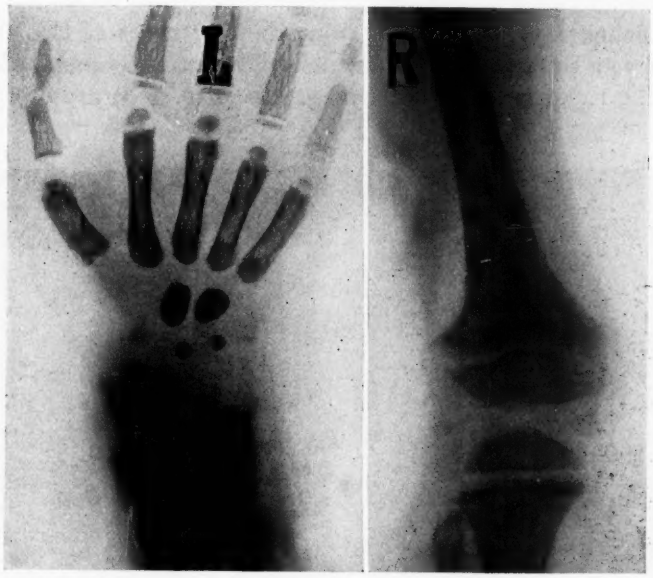


FIG. 111.—Renal rickets in a boy, age 5½ years, showing marked changes at epiphyseal line.

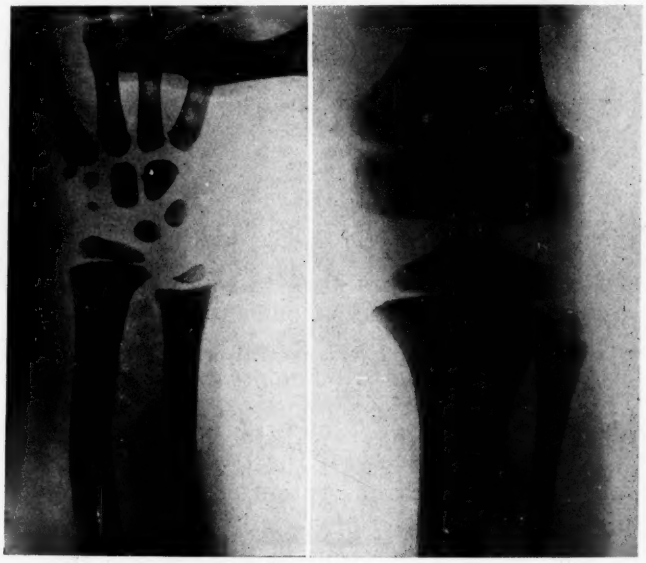

FIG. 112.—Same case as *Fig.* 111 three years later, showing epiphyseal changes healed.

or complete, near the ends of the diaphyses of the chief bones of the limbs. Even when complete they may be remarkably painless. There is always a considerable piece of metaphysis left on the epiphyseal side of the fracture. Syphilis is the only disease I know of which gives rise to softening and yielding at this particular point in several bones. Are the changes secondary to the nephritis, or are they both (as seems more likely) the result of a common infective cause ?

**Cœliac Disease.**—Judging only from the few cases of this disease I have been called upon to treat for deformities, the bone changes do not appear to be quite like rickets. The two skiagrams (*Fig.* 113) are of the same knee.

FIG. 113.—Cœliac disease in a girl. Skiagrams of right knee-joint taken at ages of 7 and 17 years respectively.

They were taken at the ages of 7 and 17 respectively. Are these bone changes the result of the disease or of the treatment ? Are we to attribute them to the faulty digestion and absorption of fats or to the great reduction of fat in the diet ordered by the physician ? It may be noted that great weakness of the lower limbs may occur in this disease, and even loss of the knee-jerks.

## ENDOCRINE ERRORS.

*Table II* shows the main types of disordered growth that result from disturbance of internal secretion so far as our present knowledge goes. It is compiled from the writings of Tidy,[21] Garrod,[22] and Gilford[23] respectively. It is impossible to enter into a discussion of all these various conditions, but a word or two may be said about some of the less known types. The Lorain

*Table II.*—General Disorders of the Skeleton due to Endocrine Disturbance.

| Gland Affected | Type of Disturbance | | Result |
|---|---|---|---|
| *Pituitary* Anterior lobe | (1) Over-secretion | (*a*) Before growth ceases .. | Gigantism |
| | | (*b*) After growth ceases .. | Acromegaly |
| | (2) Under-secretion | (*a*) Before puberty .. .. | Lorain type of infantilism |
| | | (*b*) After puberty .. .. | Nil |
| Posterior lobe | (1) Over-secretion .. .. .. .. .. | | Nil |
| | (2) Under-secretion .. .. .. .. .. | | Diabetes insipidus |
| Whole gland | Under-secretion .. .. .. .. .. | | Fröhlich's syndrome |
| *Thyroid* .. | Under-secretion .. .. .. .. .. | | 1. Cretin<br>2. Brissaud's type |
| *Suprarenal* .. | Under-secretion .. .. .. .. .. | | Infantilism (? mollities ossium) |
| *Pineal* .. | Under-secretion .. .. .. .. .. | | Gigantism plus precocious sexual development |
| *Ovaries* .. | .. .. .. .. .. .. .. .. | | Osteomalacia |
| *Unknown cause* | .. .. .. .. .. .. .. .. | | Progeria |

type suggests a small adult, is not fat, and is sexually infantile. The limbs and bones are slim. The epiphyses do not unite, or only do so very late. The Brissaud type of infantilism, on the other hand, displays a round fullness of the soft parts and a large head. All the types mentioned, except those associated with gigantism, are commonly, though not invariably, connected with dwarfism. Progeria is the name given by Gilford to a dwarf displaying a mixture of infantilism and senility. The inclusion of a suprarenal type depends on the fact that certain cases have shown remarkable improvement on the administration of suprarenal extract.

**Mollities Ossium and Osteomalacia.**—These are so rare in this country that it has been found almost impossible to study them. An occasional radiogram has convinced us that there is a disease of childhood in which the pelvis

and other bones are unduly soft, so that they yield to the normal pressure which they should be able to resist, without there being any signs which warrant a diagnosis of rickets, osteogenesis imperfecta, or fibrocystic disease. It is interesting to note that cases of this disease, mollities ossium, which is an acquired and sometimes a painful affection, have been cured by the administration of adrenalin. As to osteomalacia, it would seem that in India, where it is common, it is almost inextricably mixed up with late rickets (*see* Hutchison and Miss Stapleton[24]). The great rarity of the disease in this

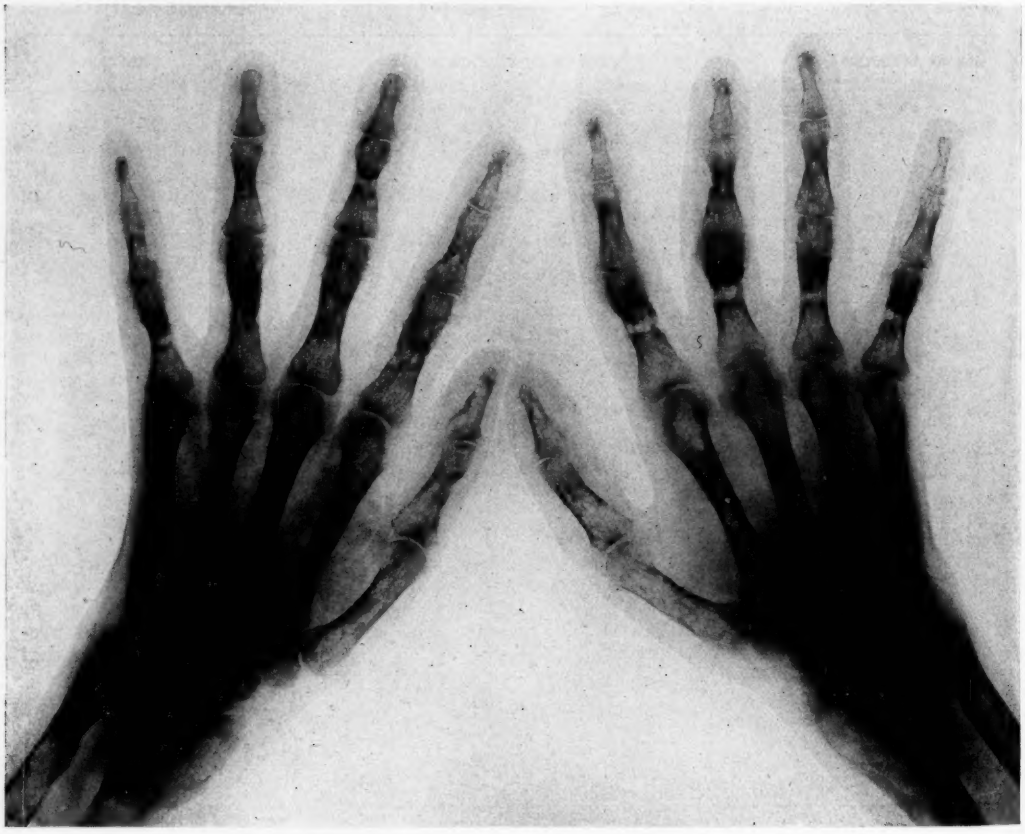

FIG. 114.—Osteomalacia. Hands of a woman, age 46 years.

country warrants including a skiagram of the only adult case I have had the opportunity of examining. The subject was a woman of 46 who had had nine children. The disease began with swelling and pain in the feet when carrying the last but one child, eight and a half years before she was seen. She never got well after this pregnancy, and symptoms came on again with the last pregnancy seven years ago, with pain and stiffness in the legs and spine. Recently she had been losing the use of her hands, and these swelled if she tried to use them much. An attack of bronchitis three months ago had made her worse, and she had been unable to walk since. The skiagrams show general abnormal translucency of practically all the bones, with thinning of the cortex,

the typical deformity of the pelvis, and very curious irregular decalcification in the hands (*Fig.* 114). The phalanges, which show a clear band in the shaft adjacent to the irregular band of increased density, look as if they could be fractured with ease. There is some loss of definition of the joints. The essential change in this disease would seem to be a halisteresis, or simple decalcification, though according to Dawson and Struthers[25] there is later some " bone apposition which remains osteoid during the period of the disease ".

## AFFECTIONS OF UNKNOWN ORIGIN.

**Osteitis Deformans.**—Of the affections of which the cause is unknown, we can only refer to one, osteitis deformans, which is of interest in connection with what has been said of striation of the bones and dyschondroplasia. Thurstan Holland[26] has called attention to the curious striated appearance of some of the bones in osteitis deformans when they themselves are not affected by the disease, and says these changes are probably some of the earliest in the disease (*Fig.* 115). A foot may show very marked striation when the tibia of the same leg shows the typical changes of osteitis deformans.

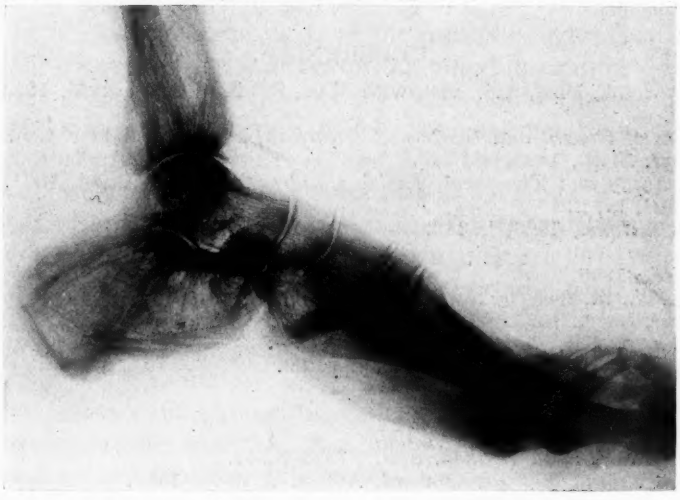

FIG. 115.—Osteitis deformans. Woman of 70 years. Foot showing striation of bones.

It is often said that the tibia is lengthened in this disease. This is only true in a sense, in that the affected bone takes a curved course instead of a straight one, but the distance between the knee and ankle is not increased; if anything it is diminished. I cannot agree with those who say that no distinction radiographically can be made between osteitis deformans and fibrocystic disease, though I admit the existence of many border-line cases. The connection of the parathyroids with this and other allied affections I cannot deal with. I will only say that so far as Paget's disease is concerned I cannot as yet discard the infective theory. Before leaving this subject may I refer to a letter I received from the late Dr. Robert Lovett shortly before his death.

In the letter he mentions some experiments he was interested in at the time, in which monkeys were fed on vitamin-free diets. One of these monkeys developed typical Paget's disease. He then discovered that three monkeys in the Philadelphia Zoo had developed Paget's disease. Dr. Clark, who reported this occurrence, suggested that osteitis deformans might be a deficiency disease, at one stage at any rate.

The subject of this paper is so immense that one has found it hard to know where to begin and harder still when to stop. The breadth of the subject is the only excuse for what must seem but a disjointed series of observations. Instead of clearing the air, these few remarks may only have added to the fog. If we have managed to interest you by a point here and there, and, perhaps, to stimulate some of the members to devote themselves to the study of the difficult problems with which the subject abounds, we shall feel that our poor efforts have not been entirely in vain. In conclusion an expression of deep gratitude is offered to the colleagues and others who have permitted such free access to their cases, and particularly to Dr. Bertram Shires, who has been responsible for the majority of the radiograms.

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# A CASE OF EFFUSION INTO THE LESSER SAC OF THE PERITONEUM ASSOCIATED WITH GALL-STONES, AND TREATED BY POSTERIOR DRAINAGE.

By GEORGE T. MOWAT, Glasgow.

Our knowledge of the pathology of cystic conditions in the neighbourhood of the lesser sac is necessarily uncertain, because these cases are, as a rule, amenable to the simple treatment of drainage, and, as the mortality is small, the number of pathological investigations which can be carried out is restricted. Meanwhile the classification is far from satisfactory, the attempt to bring all these conditions under the definition of 'true and false cysts of the pancreas' being one which is only justified by lack of more detailed knowledge on the subject. That in a large number of cases these cysts are of pancreatic origin there can be little doubt. True cysts of the pancreas, retention or otherwise, effusions into the lesser sac from pancreatic inflammation or injury, must account for the majority of cases; but there is also little doubt that accumulations of fluid in the lesser sac may occur in which the pancreas plays the rôle of an interested but inoffensive neutral. Jordan Lloyd showed that clinically these accumulations closely resemble true cysts of the pancreas of large size. Opie has shown that true pancreatic cysts are lined with epithelium; the retro-peritoneal pseudo-cysts due to injury of the pancreas are lined with connective tissue; while effusions into the lesser sac must necessarily be lined by peritoneal membrane. With this in view, examination of the lining and excision of a small piece of the wall of the sac for microscopical examination should be of help in diagnosis.

While the presence of pancreatic enzymes in the fluid in the lesser sac definitely incriminates the pancreas, it is more difficult in the absence of those enzymes to exclude the pancreas altogether as an exciting cause. The following is a case of effusion into the lesser sac associated with gall-stones and inflammatory adhesions around the cystic duct and the foramen of Winslow, where the pancreas was found to be normal and there was evidence of the irritation being a direct spread from the biliary passages.

M. R. was a married woman of 40, and had had no previous illnesses. In February, 1926, she began to have periodic attacks of vomiting, which had no relation to the taking of food. Later, vomiting became more frequent, though still without relation to food, until, at the time of her admission into hospital in April, 1926, she was vomiting at least once daily. From March, 1926, up till the time of admission, she complained of a fixed discomfort in the epigastrium, chiefly on the left side and unaffected by intake of food or by vomiting. She also noticed that her waist was becoming larger. There was nothing in her history, past or present, suggesting biliary or pancreatic disease.

For the past three months she had been growing more constipated and was losing weight rapidly.

On Admission.—She was admitted to hospital on April 5, 1926, pale, anæmic, and with a visible rounded enlargement of the upper abdomen. On examination there was tenderness on pressure over the left rectus region of the epigastrium, and a smooth, tense, rounded mass was found to occupy the upper part of the abdomen. Its limits were as follows: To the right, it ended in a rounded border at the outer edge of the rectus muscle; to the left, it filled the whole of the left hypochondrium and part of the left lumbar region: below, it extended to 2 in. below the umbilicus, where a smooth, curved margin ran from the outer border of the right rectus to the loin; above, it disappeared below the costal margin. The limits seemed to be typical of an accumulation of fluid in the lesser sac of the peritoneum. No tenderness was present over the gall-bladder, and the tests for pancreatic inefficiency were all negative.

Operation.—On April 7 she developed thrombosis of the left femoral vein, and it was not until April 19 that operation could be undertaken. A median incision was made in the epigastrium, and the tumour, about the size of a Rugby football, exposed. The stomach ran round its convexity in the form of a flattened band, 1½ in. wide, while the transverse colon was attached along its lower border. Above, it extended behind the liver to the vault of the diaphragm, and, to the left, it filled the left hypochondrium and lumbar regions. A cannula was inserted through the gastro-hepatic omentum, which was 6 in. in length, and four pints of clear serous fluid were evacuated. The opening was then enlarged and the cavity was found to be the lesser sac, lined with peritoneal membrane. The pancreas to all appearance was normal, and no induration or evidence of chronic pancreatitis was found. The stomach and transverse colon were free from disease. Examination of the biliary tract revealed a small thickened gall-bladder bound down to the liver with dense pearly-white adhesions. The same type of adhesion was present round the cystic duct, and was well-marked in the region of the foramen of Winslow, which was closed. The gall-bladder was opened and about thirty stones removed from both it and the cystic and common bile-ducts. The gall-bladder was drained and the opening of the gastro-hepatic omentum was sutured. Examination of the bile showed it to be sterile, and the gall-stones were composed entirely of crystals of lecithin and cholesterin, also sterile. The fluid from the sac was sterile, free from pancreatic enzymes, and of the composition of ordinary serous exudate. Three weeks later the abdominal wound was healed, but the sac was filling up again. On May 19 a 5-in. incision was made, running outwards parallel to and ½ in. below the twelfth rib, on the left side. It began at the outer border of the erector spinæ, and extended outwards and slightly upwards. The latissimus dorsi and external oblique muscles were cut superficially, and the tendinous interval between the quadratus lumborum and the internal oblique and transversalis muscles was sought. This was incised, and the internal oblique and transversalis muscles were cut in a forward and slightly upward direction. Only the transversalis fascia, as it ran inwards to cover the quadratus lumborum, now intervened. This was incised in the direction of the wound, and the last rib was pulled upwards

with a broad retractor. The spleen was displaced forwards, and the kidney backwards, when the lieno-renal ligament bulged downwards into view. The lesser sac was incised through this, and a tube of 1 in. diameter was inserted.

Convalescence was prolonged until September by thrombosis of the right femoral vein, and by a small sinus which persisted for over two months. When seen in January, 1927, both wounds were soundly healed, and there has been no recurrence of fluid in the lesser sac. She is well and is putting on weight rapidly.

## SUMMARY.

1. The fluid was definitely contained in a sac, the wall of which was lined with peritoneal membrane, and which conformed to the usual boundaries of the lesser sac. It was serous fluid containing no organisms or pancreatic enzymes.

2. The pancreas was uniformly normal; there were no patches of thickening, no cysts, nor anything to suggest a pancreatitis secondary to the biliary condition.

3. The presence of dense adhesions around the upper end of the common bile-duct, blocking the foramen of Winslow, and unassociated with any symptoms, suggests a low type of inflammatory condition which spread directly to the lining of the lesser sac, giving rise to slow effusion.

4. While marsupialization of a true pancreatic cyst is an efficient method of treatment, an accumulation of fluid in the lesser sac presents a different problem. The irregular shape of the sac, with the long subphrenic pouch, extending behind the liver, makes drainage a less simple matter, and the infection which follows marsupialization gives rise to the unpleasant prospect of a posterior subphrenic abscess. Drainage through the anterior abdominal wall, while efficient in the smooth, round, true pancreatic cyst, is less likely to be so efficient in lesser sac effusions of irregular shape, and the posterior incision affords, in the semi-recumbent position, a drainage at its most dependent part, the posterior surface low down and to the left. Anterior drainage, unless the patient is kept in the prone position, is of the overflow type, and, for obvious reasons, must be less efficient than drainage posteriorly, which is easily done and gives all the requirements of a true drainage.

# *VISITS TO SURGICAL CLINICS AT HOME AND ABROAD.*

## THE SURGICAL CLINICS AND HOSPITALS OF MADRID.

THE medical world of Madrid is summed up to the average British medical man in the world-renowned name of Ramon y Cajal. There is, however, much activity going on, and, so far as surgery is concerned, a band of workers is busy in Madrid who, with the modern hospitals at present under construction, will rapidly bring Spanish surgery into the front rank of their European and American colleagues.

It may have been the language difficulty which has prevented the British surgeons visiting the Madrid clinics in the past; they can rest assured that this difficulty is compensated by the welcome they will receive, and that surgery, like all true art, is universal, and needs very little language which is not common to all its votaries. The interest of their Majesties the King and Queen of Spain in hospitals, and indeed in everything which affects the welfare of their people, is well known. Their relationship alone with Great Britain would assure the British surgeon of a warm welcome in the surgical clinics of Spain. Such was the case, indeed, when recently a number of British surgeons spent a week in Madrid. They visited the various hospitals—old, new, and those under construction; the various pathological institutes; also institutes for re-education of the injured. These latter are not only hospitals for the treatment and recovery from immediate injuries, but also institutes in which an injured man recovers that peace of mind which assures him that, though injured and in some sense crippled, he is not altogether an odd lot in the general labour market. In this respect the hospital authorities of Great Britain might learn much from their Spanish colleagues; but the whole question will be gone into more fully later.

The old hospitals of Madrid are naturally situated in the older quarters of the town. The newer hospitals and those under construction are on the outskirts of the city and in the neighbourhood of the proposed University city, the realization of which is so near to the heart of the King and Queen. The authorities are adopting the course of building new modern hospitals on beautiful sites, where there is ample room for expansion on the pavilion system, rather than making abortive efforts to remodel old buildings on already overcrowded sites.

They have evidently given up the idea, which is now so truly old-fashioned, that it is necessary for a hospital to be situated in the midst of its clientèle. Where is the clientèle of a modern hospital situated nowadays? A small hospital of the nature of a casualty clearing station will meet all immediate emergency requirements.

**The Princessa Hospital.**—On Monday, April 19, three clinics were visited at the Princessa Hospital (*Fig.* 116), namely, those of Dr. Cifuentes, Dr. Blanc Fortacin, and Dr. Slocker. The Hospital is a State hospital containing 350 beds; those patients who can afford to pay towards their maintenance are expected to do so; the sum is about ten pesetas a day (i.e., fifty shillings a week).

Suprapubic Cystostomy.—Dr. Cifuentes is the urologist. The operation seen was a suprapubic cystostomy for drainage, as a preliminary to removal of the prostate at a subsequent date. The preliminary investigation of the patient had been very thorough. A regular printed questionnaire is used for each case, profusely illustrated with diagrams of the genito-urinary system, so bringing home forcibly to the student that the system must be considered as a whole, rather than as a series of independent organs. The skiagram of the genito-urinary system of the case was negative.

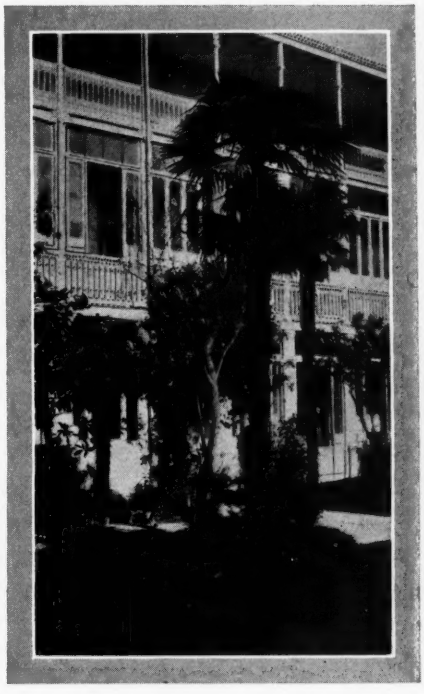

Fig. 116.—The Princessa Hospital, Madrid.

*Operation* 9 *to* 9.30. The head and feet of the patient were both lowered so as to make the lower abdomen prominent. Local anæsthesia was used—0·5 per cent novocain. After infiltration the skin was incised longitudinally. The sheath of the rectus was then infiltrated, opened, and the wall of the bladder exposed. This was then infiltrated with novocain. Two silk ligatures were passed through the bladder to act as tractors, and the bladder was opened longitudinally between them.

To the surprise of the operator, assistants, and onlookers, twelve stones were present in the bladder, the largest the size of a pigeon's egg, the smallest that of a marble. The stones were uric acid. The bladder was drained with a wide-bored Marianne's tube, with a small tube attached laterally for lavage.

A series of skiagrams was demonstrated illustrating the use of pyelography for distinguishing kidney tumours from other abdominal tumours, especially the tumours of the spleen.

Dr. Blanc Fortacin is the gynæcologist; he did a supravaginal hysterectomy for a large fibroid.

Supravaginal Hysterectomy.—*Operation* 9.55 *to* 10.40. The anæsthesia was intrathecal by tropacocaine. Catgut was used throughout for ligatures. The cervical canal was sterilized with the actual cautery and not closed.

Dr. Slocker specializes in abdominal surgery, especially that of the stomach. We saw him do two operations.

1. Posterior Gastrojejunostomy for Duodenal Ulcer.—*Operation* 10.45 *to* 11.5. The anæsthesia was by closed ether. The abdomen was opened

in the middle line and the lesion demonstrated. The usual posterior no-loop gastrojejunostomy was done, except that three layers of sutures were inserted both anteriorly and posteriorly, all of catgut. The first layer took the serous coat only, the second the seromuscular coat, this being put in before the mucous membrane was opened. Clamps were used both for the stomach and jejunum, and straight needles. The abdomen was not otherwise explored. The abdominal wall was closed in layers, the skin being united with Michel clips.

2. Supravaginal Hysterectomy for Fibroid.—*Operation* 11.20 *to* 12.5. Intrathecal anæsthesia by tropacocaine was used. The case presented great

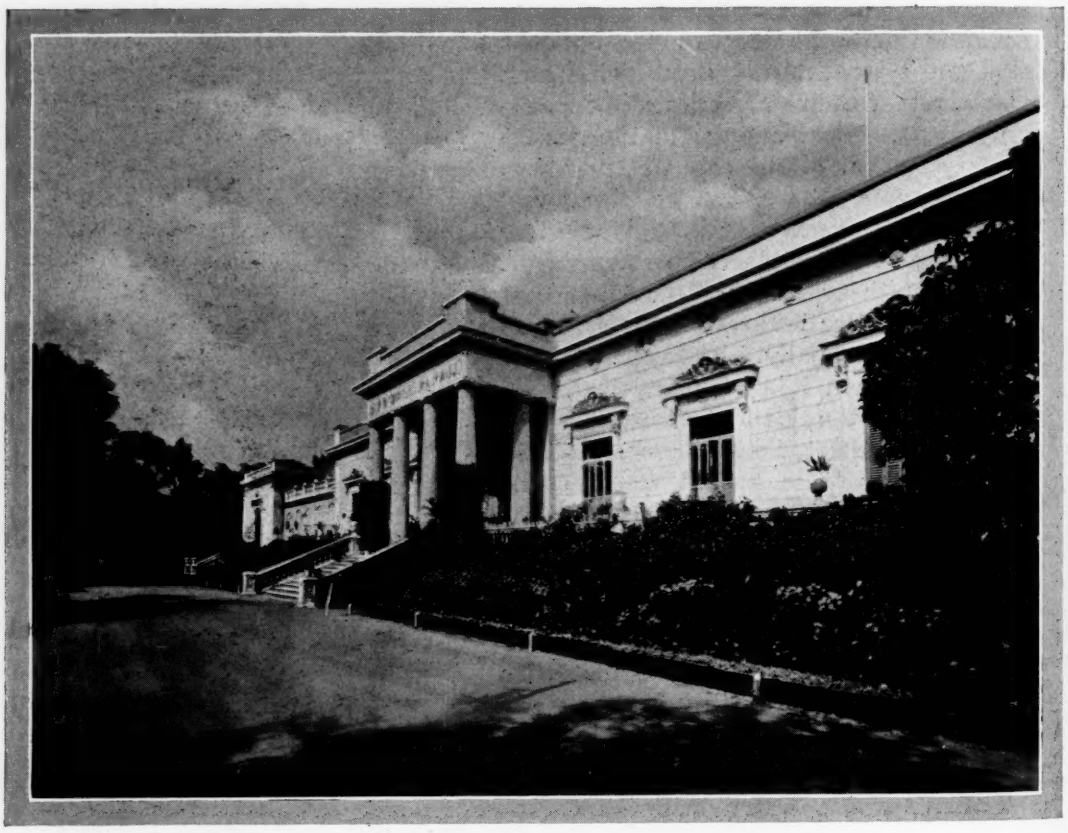

Fig. 117.—The Orthopædic Hospital and Institute, Madrid. Front view.

technical difficulties owing to deformity of the pelvis and the presence of a double pyosalpinx. The cervical canal was closed with thick catgut. An omental graft was placed over the sigmoid colon where the pyosalpinx had been adherent. Faure's needle was used for the deep pelvic work. The abdominal wall was closed in layers.

**The Orthopædic Hospital and Institute,** which we are enabled to illustrate in *Figs.* 117–120, for re-education of the injured, is situated on the

outskirts of Madrid. The literal translation of the address is 'beautiful view', and the situation deserves it. The work done is excellent. We were conducted round in a most thorough and courteous manner by the director, Dr. Oller.

The building might be described as a new old one. It was built in 1816, and for many years was a private house belonging to the Marquis of Salamanca; later it was used as a workhouse, and indeed a part of the building is still occupied by the indigent aged. For the last four years, much enlarged and brought up to date, it has been used for its present purpose.

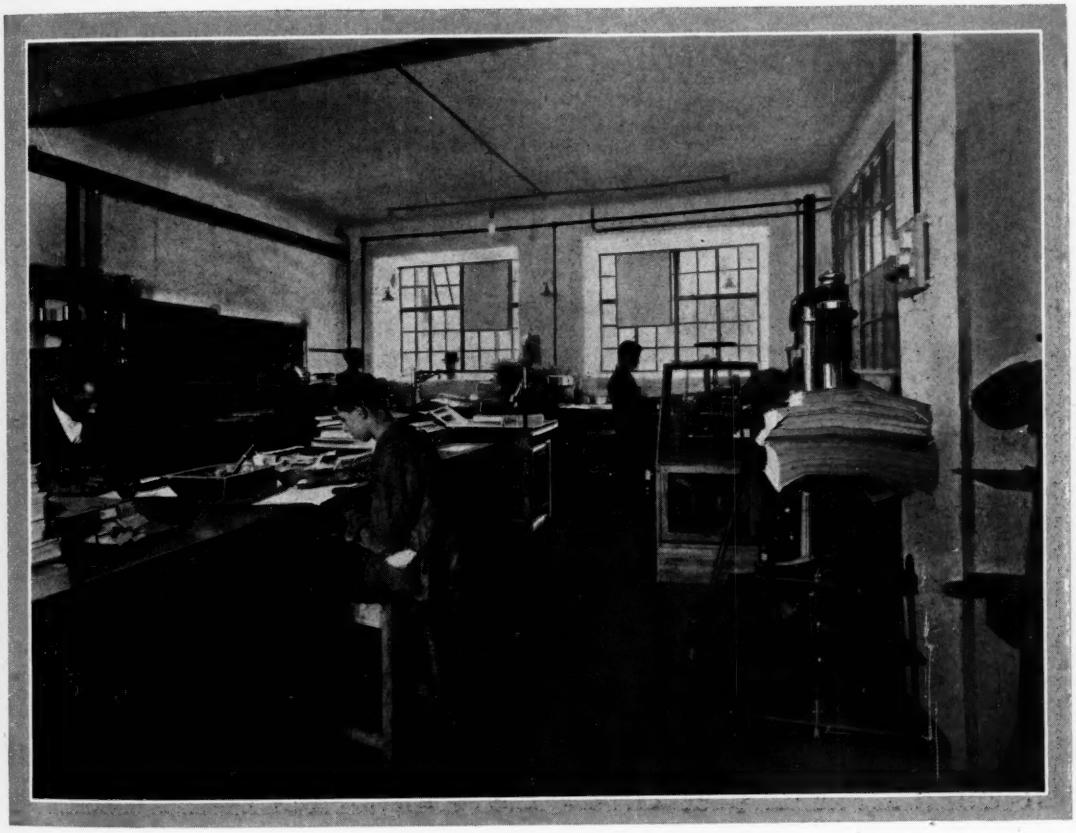

FIG. 118.—The bookbinders' workshop.

There are 250 beds, complete with X-ray department, suite of rooms for operations, massage, etc.

It receives patients from all parts of Spain: locally, often immediately after the accident; more often those from a distance only months, or even years, after the injury. The cases, therefore, resolve themselves into the two classes so well known in our own country—those who have never been allowed to drift away from expert treatment, and those who have often by chance drifted towards it, perhaps too late for anything to be done. It must therefore be judged by its successes rather than condemned by its failures.

We visited first the wards and then the library. The latter is decorated in the Moorish style of the Alhambra, the coolness and shade being very necessary. All the representative surgical journals seem to be on the shelves. The books were evidently being chosen with great care, and there would be very few surgical subjects which could not be readily referred to.

We next visited the theatre, where plays are given both for and by the patients; they also have cinematograph entertainments and lectures. The director rightly considers this a very important asset to promote the recovery of the patients.

FIG. 119.—The library.

We were then shown round the school. One of the main difficulties with which the director has to contend is the illiteracy of many of the patients. Here the illiterate are taught to read and write, and the literate taught the essentials of mechanics, drawing, etc., so that they can be fitted for one of the main objects of the Institute, namely, the re-education of the patient in some trade or calling by which he has a fair chance to keep himself by getting work in the general labour market.

The finances of the Institute are supported by the Ministry of Labour, which pays at least 400,000 pesetas a year (about £16,000) towards the

upkeep of the Hospital. Everything made in the Hospital is sold, and this brings in a further 60,000 pesetas a year (about £2400). In addition, the patients, if possible, pay a small amount towards their maintenance. Apparently the works at which the man was injured do not have to pay towards his maintenance in hospital, though the injured man does receive compensation up to 35 pesetas a week (27s.) towards the maintenance of himself and his family. There is, however, nothing comparable with our schemes of workmen's contributions to the hospitals, nor contributions by employers, because most are State-aided hospitals.

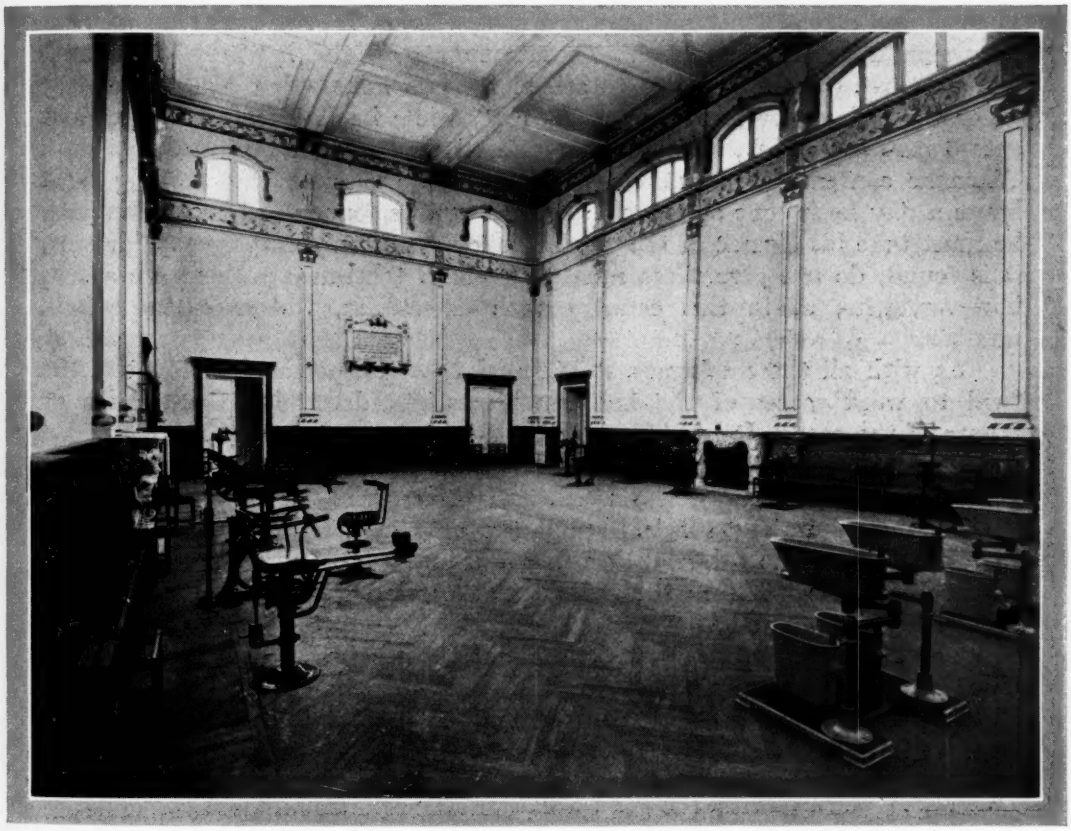

FIG. 120.—The hall for mechano-therapy.

The Institute is fitted with a very complete remedial exercise and gymnastic department attached to the massage and electrical departments. The director is a great believer in the 'Zander' method of remedial exercises.

The workshops for instructional purposes were under the charge of an overseer who spoke excellent English and had been employed as an engineer by Messrs. Vickers & Co. Ltd. in England for many years. He has to make a personal study of each man so as to form an idea of what type of work he is fitted for, and also whether or not it is quite impossible for him to do his old work after suitable re-education. Naturally as far as possible a man is put to his own or some kindred trade. In some cases this is quite out of the

question. He then has to be completely retrained. The occupations for which he can be re-educated include boot making and repairing, fitting, milling, wireless work, electrical work, wood-working, furniture making, leather working, book-keeping, etc.

The wood-working and furniture-making departments appeared very complete and the instruction very thorough. The men are first taught how to use their tools, then to make models of the proposed furniture, and finally make the ordinary pieces; some shown were excellent in make and quality.

Many of the patients being trained are not in-patients, but live in the vicinity and attend for treatment and instruction. It is often necessary that they should go to the school. These men attend the Institute at 8 a.m., and on three days a week go to school for two hours each day and on the other three days for four hours. At first, while he is learning, the patient gets 6 pesetas a week, and this is advanced gradually according to his skill. When he can earn 44 pesetas a week (about 30 shillings) by working eight hours a day he is sent out to a job if one can be found for him; otherwise, he must take his chance in the general labour morket. Employers of labour, it is found, do not give these men preferential treatment; there are a few, however, who, as in this country with ex-soldiers, will accept a certain number.

As with all these schemes, it is found that the personal factor, and the 'will to work' asset of the individual member, determine the success or failure of the case. It has been found that the time necessary for retraining varies considerably—from a few months in a case of boot-repairing to two years for electrical or wireless work, and three years for book-keeping.

All the splints and surgical appliances used in the Institute are made in the workshops. The artificial limbs for the lower extremity above the knee-joint sell for about £17 in the general market, and appeared to be very well-constructed, light, and efficient.

There was an air of camaraderie about the whole Institute; all the men observed at work were obviously trying to do their best in spite of their disability. The atmosphere of competition, so to speak, seemed to have improved the morale of the men.

One knows of no similar hospital in this country except the tuberculosis colonies of Papworth and other similar places. In these latter, however, it is assumed that the man will never be able to take his chance in the general labour market. He is trained to regard himself as an 'odd lot'. In this Institute the men are encouraged not to regard themselves as in any way an 'odd lot', but to look forward to the day when they can compete on level terms with their fellow-men so far as the quality, efficiency, and speed of their work is concerned.

**The New Military Hospital** of 2000 beds in 30 pavilions is also situated on the outskirts of Madrid. Transport is so organized that the wounded are in this hospital within three days from the line in Africa. It is complete with surgical, medical, ophthalmic, aural, infectious, and dermatological sections. It has a very complete pathological and operation suite, and

remedial and re-education departments. The ideal of the Hospital is summed up in the statue that meets the visitor on his entrance—a wounded soldier breaking his crutches across his knees—exquisitely executed in bronze.

We were conducted round by the officer commanding and the chief of the surgical section, Dr. Bastos. The cases shown bore witness to the efficiency of the work. There were especially two cases of cineplastic amputation of the arm. In one, a man with the arm amputated just below the shoulder-joint, the artificial hand could be opened and shut with ease, also small objects picked up and held between the thumb and forefinger.

The cases of paraplegia from gunshot wound of the spinal cord with retention of urine were being treated by leaving alone and expressing the bladder contents by hand every two hours, until automatic control was established. No catheterization or suprapubic drainage was used.

**The University Surgical Clinic of Madrid at the San Carlos Hospital.**—This is a large general hospital, and takes as in-patients not only accidents and those cases admitted in the ordinary way from all over Spain, but any case admitted to the other hospitals in Madrid, which is thought to be of value for students, can be transferred there. The Hospital is closed down for three months during the hot weather and summer vacation, and the cases remaining are distributed into the other hospitals of the city.

The surgical clinic of Professor Doctor Cardinal is a busy one, judging by the list of operations for Wednesday, April 20. There were three gastric cases diagnosed gastric ulcer, two cases of appendicitis, one of cholecystitis, and seven hernias. These, with a hæmorrhoid case, made up a list of fourteen operations to be done between 9 a.m. and noon.

Three tables were in use at the same time, and the work was silently, efficiently, and deftly performed.

1. Cancer of Pylorus (Gastrectomy by Professor Cardinal).—*Operation* 9.30 *to* 10.25. The anæsthesia was pure chloroform administered by the drop method. The skin was cleaned by ether, and then iodine was used. The incision was made through the middle line. The greater curvature of the stomach was first freed, then the lesser curvature, and the stomach then divided. The duodenum was clamped with crushing forceps, and the crushed area tied tightly with silk. A purse-string suture of silk was passed round the duodenum distal to the silk ligature. The duodenum was divided by the actual cautery, and the cut end invaginated. The raw surface of the pancreas was shut off with an omental graft. The union of jejunum to stomach was by the posterior Polya method, proximal end to greater curvature, the intestine being opened by the actual cautery.

The ligature and suture material was silk throughout; the suture line of the gut was always begun in the middle, so that the knots were never at the periphery. The abdominal wall was closed in layers, silk being the suture material used.

2. Operation for Cholecystitis (Professor Cardinal).—*Operation* 10.25 10.45. The anæsthesia was by pure chloroform administered by the drop method. No gall-stone pillow was used, nor any rest to push the upper

abdomen forward. The dissection of the gall-bladder from the liver was done from above downwards. The cystic artery and duct were tied in one catgut ligature, the duct being divided with the actual cautery. The abdomen was closed with no drainage.

The appendicectomies were done through gridiron incisions. The hernias were done by the Kocher method of treating the sac.

Professor Olivarez did three operations :—

1. For a Duodenal Ulcer in a man 39 years old. He had a typical history extending over twelve years, and recently had had symptoms of pyloric stenosis superimposed. The X ray showed a dilated stomach with delay in emptying. The operation done was that of partial gastrectomy. *Operation* 11.5 *to* 12.15. The anæsthetic was ether given by an inhaler. The stomach was first separated along the greater curvature. De Martel's clamps were used both for the stomach and duodenum. The jejunum was joined to the stomach by the posterior Polya method, the proximal end of the jejunum being put to the lesser curvature.

2. For Hydatid Cyst of the Liver.—*Operation* 12.25 *to* 12.50. The anæsthetic was ether given by an inhaler. The incision was made through the rectus, the cyst exposed, and the surface moistened with 5 per cent formalin. The cyst was then aspirated and 5 per cent formalin injected. After being emptied, the cyst was marsupialized and drained.

3. Cholecystectomy for Gall-stones.—*Operation* 12.55 *to* 1.15. The anæsthetic was ether given by an inhaler. The incision was made through the rectus. The gall-bladder, which was firmly adherent to the stomach, was dissected free. The cystic duct was defined, tied, and divided. The gall-bladder was then removed. A tube was placed down to the stump of the duct. The abdomen in all these cases was closed in layers, catgut being used for the deeper ones.

Professor Don Sebastian Recasens, the Dean of the Medical Faculty of Madrid, is the professor of gynæcology at the University. It was entirely due to his unfailing energy and courtesy that the visitors were enabled to see so much of the work in the surgical clinics. He performed three operations :—

1. For Diseased Appendages.—The case had a long history of pain and discomfort. He explored the pelvis through the usual mid-line incision. He removed the left ovary and tube, but was able to save the right. He had to deal with a considerable raw area on the pelvic colon. He removed some omentum, had it melted down in the theatre, and poured the liquid fat over the raw area. He stated that he had been doing this for a number of years, and thought it was the surest way to prevent the adhesions re-forming.

2. Myomectomy for Fibroid of the Uterus.—The case presented no unusual feature. The Professor stated that he considered myomectomy the operation of choice.

3. Hysterectomy for Fibrosis of the Uterus with Menorrhagia.—The abdomen was closed in all these cases in layers, using catgut for the deep and silkworm gut for the superficial layer.

**The Medical School.**—The motto of the Medical School of Madrid is "Nunquam sine anatomica artem chirurgicam possidebis", and the course

for the medical student supports this, as more stress is laid upon anatomy than physiology.

The medical student usually 'enters' on his course at the age of 16 years. The first year is spent in the Faculty of Science doing chemistry and physics. At the end of this year, and after passing the necessary examination, he enters the Faculty of Medicine. The course in the Faculty of Medicine lasts six years. The first year is given up entirely to anatomy, doing the usual dissections and attending lectures. He also does his course of microscopical anatomy this year under the direction of the famous Professor Ramon y Cajal.

The second year is spent doing anatomical dissections and physiology, both practical and lectures. The third year is spent at pathology, general and special; also attending a course of therapeutics, both lectures and practical laboratory work. During the fourth year he attends classes of instruction in clinical methods. The fifth and sixth years are given up entirely to clinical work. During the fifth year he attends a course and actually does operations on the cadaver, a course described as topographical anatomy. There is an examination at the end of each year which must be passed before he is allowed to proceed to the next year's work.

There are three professors of general surgery in the University of Madrid; they incline, however, to specialize—e.g., one is interested mainly in urology, another in abdominal surgery, and a third in the surgery of fractures and the cranium.

Each professor has six whole-time assistants. The assistants are appointed after open competitive examination, and each step of the interne's career is dependent upon passing competitive examinations. The professors also have to sit for an examination organized by the Ministry of Education before being appointed to the chair. All so-called honorary and professorial appointments are made only after competitive and public examination. We did not ascertain if there is a time limit for a professor as regards age.

There are about 3000 medical students in Madrid University. There are also about thirteen other Universities in Spain having a medical faculty and students attending the courses. So far as we could understand, all the medical education is free; only the examination fees have to be paid by the student.

There is nothing comparable to the National Health Insurance Act in Spain so far as regards the prospects of practice for these students after they are qualified. It appears that the classes of Spain are divided, so to speak, very horizontally into upper and lower classes. There does not seem to be what one might call the bourgeois element, or the lower and upper middle class.

The people all belong to large medical-aid societies to which they pay varying sums according to the benefits received, including burial. It could not be ascertained whether these medical-aid societies paid anything to the hospitals. They probably do not, as the hospitals are almost all State-aided.

In private practice the municipalities pay the doctors for the poor. The other people all belong to clubs and medical-aid societies. Practices are not bought or sold, but in every district there are private policlinics which young men are invited to join, and as years go on they draw a larger share of the profits.

**St. Adela Hospital.**—A visit was paid on Wednesday, April 20, to the Hospital of St. Adela. It is a hospital supported by the Spanish Red Cross and sometimes called the Queen's Hospital, as Her Majesty the Queen has a particular interest in it. We were shown round by the director, Dr. Victor M. Noguerras, who is justly proud of the new hospital which is under course of construction.

It is a general hospital, thoroughly equipped for surgical work only, both as regards out- and in-patients. It is the training school for lay nurses, both V.A.D. and fully trained, under the auspices of the Red Cross Society. The training for a V.A.D. is six months, for the professional nurse two years. Next year it is said that the eldest daughter of their Majesties will do the complete V.A.D. training.

**The Cancer Research Institute.**—We were conducted round the Alfonso XIII Institute for Cancer Research by the director, Dr. Joseph Goyannes. It is a new Institute, having been opened four years ago, and is situated on the outskirts of Madrid in the vicinity of the proposed new University City. There are at present 32 beds, but in two years' time it is hoped that extensions will be completed and 200 beds will be available.

This Institute, as well as being probably one of the most modern in Europe for the investigation of cancer, is a hospital completely equipped for its treatment. It is also a centre for the anti-cancer propaganda of Spain.

The State supports the Institute, paying 100,000 pesetas a year (about £4000). The patients pay 12½ pesetas a day (10s.) towards their maintenance. There is an out-patient department, open three days each week, and cases are sent to it from all over Spain.

X-ray treatment is given as a preventive after the operative treatment. In carcinoma, small and frequent dosage has been found to be more beneficial than massive doses.

**The Instituto Rubio.**—This hospital, which we next visited, contains 60 beds, and is used mainly for the training of lay nurses and as a post-graduate clinic for medical men of Madrid and district.

**The General Hospital** was visited on Friday, April 22. This is one of the old hospitals of Madrid. The wards are large and broad, some of them containing seventy-two patients in four rows. It is situated in one of the crowded quarters of Madrid, and must be an extremely busy general hospital.

Four surgeons were seen at work in their clinics. Dr. Gimenez did three operations :—

1. Gastrojejunostomy for Duodenal Ulcer.—*Operation* 9.20 *to* 9.53. The operation was of the posterior, no-loop type; silk was used for suture of the serous coats, and catgut for the mucous membrane. The abdominal incision was closed by through-and-through wire sutures.

2. Appendicectomy through a gridiron incision.

3. Removal of Epithelioma of Tongue and the glands of the submaxillary triangle of the neck on the same side. The half of the tongue was excised by the usual intrabuccal method.

Dr. Pelaez is the abdominal surgical specialist of the General Hospital. He does gastric surgery principally. We saw him do three partial gastrectomy operations for ulcers :—

1. Gastrectomy for Cancer of the Pylorus.—*Operation* 9.30 *to* 10.35. The anæsthetic was pure chloroform. The greater curvature of the stomach was first freed, then the lesser curvature. The duodenum was divided between crushing clamps, the distal end closed by passing a running silk suture over and over the clamp, and then withdrawing the clamp, pulling the suture tight at the same time. The end was then invaginated by a purse-string suture of silk. The jejunum was fixed to the stomach by the retrocolic Polya method, the proximal end of the jejunum to the greater curvature.

2. Gastrectomy for Duodenal Ulcer.—The ulcer was fixed to the pancreas. The technique was the same as in the previous case. *Operation* 10.50 *to* 11.40.

3. Gastrectomy for Gastric Ulcer on the lesser curvature. The technique was the same as in the previous case.

Hydatid Cyst.—Dr. Vigueras operated for a hydatid cyst of the liver in a girl 18 years of age. There was a history of a lump having been present for two and a half years ; no thrill was to be felt. A transrectal incision was made and the cyst exposed. Formalin 5 per cent was injected into the cyst, which was then emptied, marsupialized, and drained. The *operation time* was 11.45 *to* 12.10.

Nephrectomy and Prostatectomy.—Dr. Covisa is the urologist at the General Hospital. He did two operations—nephrectomy for calculous pyonephrosis, and prostatectomy. For the nephrectomy he used a lumbar incision. The pelvis of the kidney was very dilated, and he had some little trouble with the pedicle. The operation field was quite dry when the operation was completed. In the other case the prostate was removed at this the first operation, the prostatic bed being packed with gauze strips moistened with iodoform. The cases had been thoroughly investigated before operation had been advised.

**The Maternity Hospital of St. Maria Christina** is a new hospital, and is named after the Queen Mother, who takes a very particular interest in this hospital, and at whose instigation it was built. She gave the ground and some money towards the building, the State paying the rest of the initial expense.

It contains 150 beds ; 100 are for obstetric cases and 50 for gynæcological cases. It is also a training school for thirty midwives. There are wards in which the patients pay 6 pesetas a day (about 4/–), the fees for others ranging as high as 18 guineas a week for private rooms with bathrooms attached. These fees are for maintenance only, and do not include medical attendance.

The idea at the conception of the hospital was that it should be self-supporting. This has never been the case yet, and the Government makes up the deficit each year. It is beautifully situated, and excellently equipped with X-ray department, operation suites, pathological laboratories, lecture

rooms, ante-natal departments, etc. It is probably one of the most modern and best-equipped maternity hospitals in Europe. All the patients must be married. There appears to be no provision for the illegitimate mother.

* * * * * *

The impression left on one's mind after a visit to the hospitals of Madrid is one of progressive effort towards the attainment of a very high ideal. In Spain, as everywhere at the present time, the hospital service of the nation is evidently engaging much of the time and thought of the authorities.

In Spain it is recognized that the expense is such that the State must be directly or indirectly responsible for much of it; yet it is made clear to the people that some effort on their part is necessary to contribute to their cost of maintenance, and all pay something who can. With the interest which is being taken by the King and Queen, there is no doubt that in a very short time the hospital service of Madrid will be second to none.

The directors of all the hospitals, and the surgeons who were seen at work, are evidently much-travelled and widely-read men, who have a broad outlook, not merely upon their own little corner of medical practice but over the whole field of medical science both in the clinic and the laboratory. Very little is being left to chance, and under the guidance of the Medical Faculty of Madrid, and the energy of their indefatigable Dean, Professor Don Sebastian Recasens, the future of Spanish surgery is assured.

Anyone who wishes to visit these clinics in the future may rest assured that everything will be done to make his task easy and his visit enjoyable.

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## 'BACILLUS WELCHII CHOLECYSTITIS."

We have received the following note from the authors of the article entitled 'A Case of B. Welchii Cholecystitis' published in the April issue of the BRITISH JOURNAL OF SURGERY, p. 646.

"Our attention has been called to the omission of any reference to the case of 'Gangrenous Cholecystitis due to the Gas Bacillus' published by Dr. G. G. Cottam in *Surgery, Gynecology, and Obstetrics*, August, 1917. We regret this want of accuracy in our references the more, seeing that Dr. Cottam's case antedates by some years the two others quoted in the article, and must be considered the first recorded case of the kind in which the bacteriology was completely worked out".

# *SHORT NOTES OF RARE OR OBSCURE CASES*

## A CASE OF CARCINOMATOSIS FOLLOWING TREATMENT BY RADIUM.

By JOHN B. HUNTER,

SURGEON TO THE ROYAL NORTHERN HOSPITAL, AND ROYAL CHEST HOSPITAL, LONDON.

THE following case of carcinomatosis is of considerable interest, the secondary deposits having been treated with radium. In 1922 I reported two cases of general vascular carcinoma in which radium had been used in treating growths of the uterus. My opinion was then that in a certain number of cases radium appeared to destroy the patient's natural resistance to carcinoma, and allowed

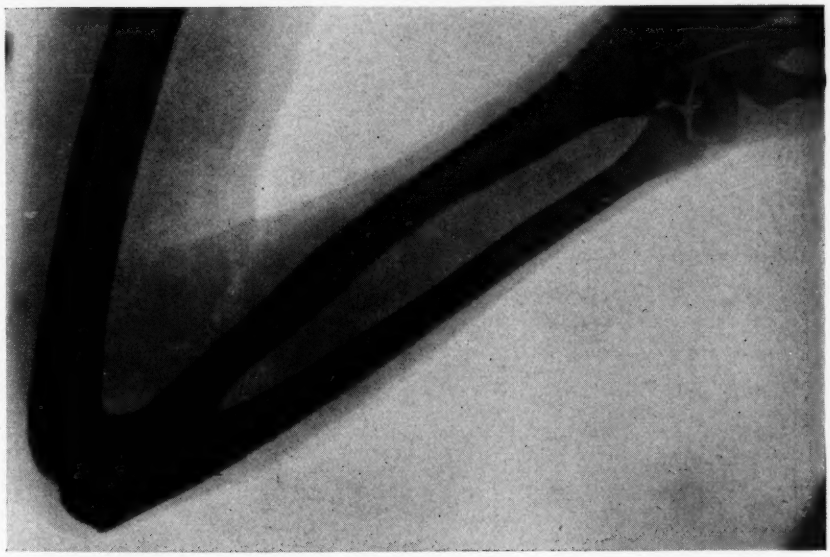

FIG. 121.—Humerus, radius, and ulna, showing growth in humerus and upper end of radius.

circulating carcinoma cells to settle, and start secondary deposits wherever they were carried. Since then I have seen a number of cases treated with radium in which the secondary deposits appeared more numerous, and in more varied positions, than in cases not so treated. This case is an extreme example. It was admitted under Dr. Batty Shaw, to whom I am indebted for permission to report it.

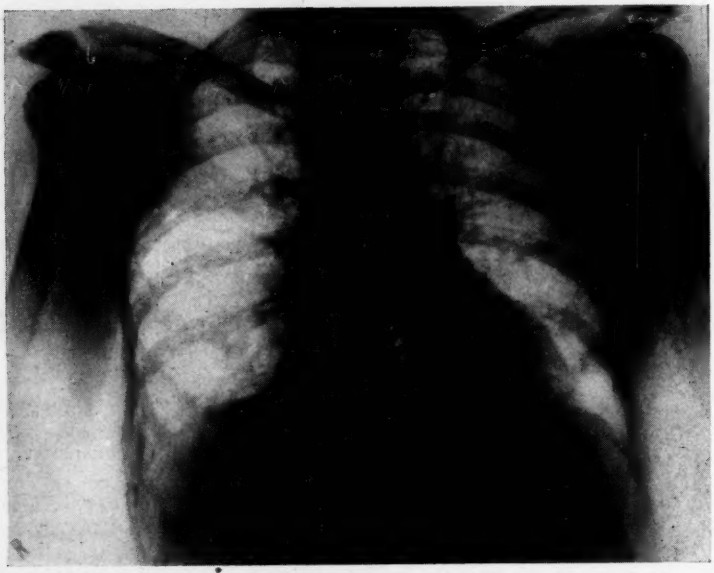

FIG. 122.—Showing condition of ribs, clavicles, and scapulæ.

FIG. 123.—The pelvis is completely infiltrated with growth.

B. C., a married woman of 43 years, was admitted to University College Hospital complaining of pains in the chest and abdomen, and glands in the neck which apparently enlarged and decreased in size. She had the scar of a radical breast operation on the right side. In both posterior triangles there were hard glands relatively discrete. On examination of the chest there were

FIG. 124.—The skull, which shows the extensive destruction of bone by the secondary deposits.

patchy areas where breath-sounds were weak. There were no physical signs in the abdomen. Her blood-count showed a secondary anæmia.

On three occasions the glands in the neck were radiated with 100 mgrm. of radium for four hours. Areas of the chest wall were also treated. The dosage and screening were as follows: (1) July 28, 1925: Inner end of left

clavicle and outer end of clavicle—100 mgrm., screened 0·5 mm. platinum, 2 mm. rubber; 4 folds lint, three hours to each place. (2) Sept. 1, 1925: Left breast region, middle of back—100 mgrm., screened as before, three hours each. (3) Dec. 18, 1925: No. 1 repeated. After each application the patient vomited. She had previously had radium treatment at the Middlesex Hospital. A skiagram of the chest showed secondary deposits in the lungs, the scapulæ, and ribs. Two months after admission she developed a nodule in the skin of the chest, and her general condition became worse. Skiagrams of the whole skeleton were taken at this time (*Figs.* 121–124), and showed secondary deposits in the skull, vertebræ, pelvis, humeri, and both femurs, as well as the situations already mentioned, but the peripheral bones appeared normal. Secondary deposits became palpable in both axillæ, the skin of the back, and the left breast. She also developed a pleural effusion on the right side, which was aspirated.

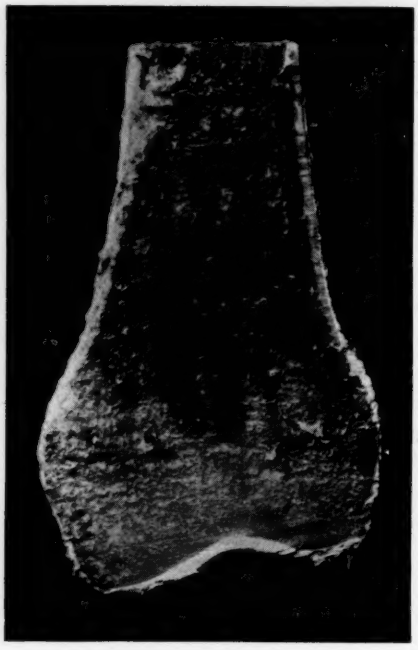

FIG. 125.—A photograph of the lower end of the right femur in longitudinal section, showing the red marrow and the secondary deposits standing out as white nodules.

The patient gradually got weaker, and died five months after admission. There was some terminal confusion, but at no time did there appear to be any involvement of the central nervous system.

POST-MORTEM FINDINGS.—The heart showed simple atrophy; there were small secondaries in the parietal pericardium. There was a bilateral pleural effusion and there were secondaries studded over the pleural surfaces, both visceral and parietal. The liver showed numerous secondaries, as did the spleen, and growth was also found in accessory spleens. There was replacement of all but a small portion of cortex of both suprarenals by firm white growth. Secondaries were also present in both ovaries and in the left breast. In the brain there was a firm large mass occupying the anterior lobe. Over the vertex, and less marked in the middle fossa and occipital region, was a thick granular sheet of carcinoma covering the bony surface of the dura. The vault of the skull was thickened and worm-eaten on its inner surface, and in two areas there was complete replacement of bone by growth.

In the other bones examined—lumbar vertebræ, ribs, right radius and femur—there was widespread secondary infiltration, and red marrow was present in the lower ends of the radius and femur (*Fig.* 125). In all cases the macroscopic presence of growth was confirmed by microscopic sections.

## A CASE OF 'WANDERING SPLEEN' CAUSING INTESTINAL OBSTRUCTION.

By H. A. H. HARRIS, London.

C. O., male, age 27, was admitted to hospital, under my care, on Dec. 14, 1926, his doctor having made a diagnosis of intestinal obstruction. The patient was of an excitable and voluble temperament, and this made the elicitation of an accurate history somewhat difficult.

History.—The patient stated that he had always had a tendency to constipation and that he habitually took aperients. Constipation had been more obstinate during the past year, while for a fortnight previous to admission there had been no satisfactory evacuation; in spite of repeated enemata, only a few small, hard masses had been passed. The last occasion upon which he had voided any fæcal material or flatus was about thirty-six hours previous to admission. He had vomited some greenish fluid on the same afternoon that he entered hospital. Pain was not a prominent feature, although there were occasional colicky attacks. There were no urinary symptoms.

Clinical Examination.—The patient, though thin, had a good colour and looked healthy. The tongue was dry and thickly coated, pulse 120, temperature and respiration-rate normal. The abdomen was slightly distended, and below and to the left of the umbilicus there was a large rounded firm mass which appeared to be rising out of the pelvis. This tumour was tender to pressure, could not be indented, and was quite immovable. The patient asserted that he had known of the presence of this lump for a year, but that it had caused him no inconvenience. Rectal examination, which caused pain, revealed a tumour in front, having a rounded margin, continuous with the abdominal swelling. The rectum was firmly compressed in its antero-posterior diameter, and the examining finger could not be passed above the free border of the tumour, nor could the mass be moved in any direction.

Three oil and turpentine enemata were given at intervals of one hour, but with no result, and the nurse reported that very little fluid could be run into the rectum. It was therefore decided that exploratory laparotomy should be performed.

Operation.—This was carried out under spinal anæsthesia combined with a preliminary injection of morphia and hyoscine. A left sub-umbilical paramedian incision was made, and on opening the peritoneum a firm, fleshy, reddish-blue mass was disclosed. At first the nature of this was not evident, but a hand passed up failed to find the spleen in its normal position, and a pedicle passing to the deep surface of the tumour was discovered. Some difficulty was experienced in delivering the spleen owing to the firmness of its impaction in the pelvis. When this was accomplished, a long pedicle, containing the tail of the pancreas, was revealed, and it was seen that it had undergone torsion through one complete turn. The gastrosplenic omentum

was intact, and the free edge of the great omentum was adherent to the viscus.

Removal of the spleen presented no difficulty. The pedicle was ligatured in several portions, distal to the tail of the pancreas, and it was found that the free ends of the vessels could then be invaginated by passing a purse-string suture through the peritoneum investing the pedicle. The abdomen was then closed without drainage.

PROGRESS.—There was no post-operative shock or vomiting, and the patient gave no anxiety for two days after the operation, during which time the bowels were evacuated. On the third day the patient vomited thrice each time about 16 oz. of light-brown material, and the pulse became rapid and thready. Examination of the abdomen at that time revealed distention and a succussion splash, and it became evident that he had an acute dilatation of the stomach. This yielded well to lavage and the prone position, and the pulse greatly improved.

FIG. 126.—Photograph of a 'wandering spleen' which became impacted in the pelvis and caused intestinal obstruction.

He remained, however, restless and talkative, and did not sleep well in spite of narcotics. On the eighth day he collapsed somewhat suddenly, the reason for which was obscure, as the abdomen was supple, the wound healed, and there was no evidence of any further gastric dilatation. He died on the morning of the ninth day. Unfortunately, permission for a post-mortem examination was refused.

PATHOLOGICAL REPORT.—I am indebted to Professor D. M. Greig, of Edinburgh, for the following pathological report:—

"*Macroscopical Appearances.*—The spleen is misshapen. The hilum is not recognizable in any hollow, but seems to be on the convex border, and the vessels are obscured by fibrous thickening. The measurements of the spleen are: length 177 mm., breadth 101 mm., thickness 114 mm., transverse circumference 355 mm., longitudinal circumference 520 mm. This

spleen has two poles, one a narrow apex and the other a blunt base; to the latter is extensively attached a piece of great omentum measuring 127 mm. in length. The vascularity of this doubtless supplemented the normal blood-supply of the spleen, which must have been much interfered with. Besides the base and apex this spleen presents three borders and three surfaces. To two of the surfaces there have been extensive peritoneal adhesions, while the third surface has remained comparatively free. Two of the borders show notches. One near the apex measures 35 mm. by 10 mm., while two notches near the base of the spleen are more or less obliterated by adhesions and organized lymph. (*Fig.* 126.)

"*Microscopical Appearances.*—This spleen shows a high degree of passive congestion. Germinal areas are almost eliminated, and there is considerable fibrosis of endothelial cells, with much necrosis."

Many authors have published cases of wandering spleen, but this is probably rather an indication of the rarity of the condition than otherwise. It may be noted, for instance, that up to September, 1920, only two spleens had been removed for this condition at the Mayo Clinic.[1] An exhaustive search of the literature has, however, failed to disclose a similar case to this in which the prolapsed organ produced intestinal obstruction by obliterating the lumen of the gut. Ledomski,[2] however, records a case in which a wandering spleen with a twisted pedicle produced an intestinal volvulus; while Finkelstein,[3] who operated on 16 cases with a mortality of 25 per cent, states that peritonitis and intestinal obstruction resulting from torsion of the spleen are 'frequent and very dangerous occurrences'. He does not, however, enter into any detail as to the mode of production of the obstruction, although he is probably referring to cases similar to that of Ledomski.

When the spleen becomes displaced it displays a pronounced tendency to drop into the pelvis, owing to the influence of gravity, and this accounts for many of the cases having been described by gynæcologists who have operated under an erroneous diagnosis of fibromyoma of the uterus or ovarian tumour. The majority of the authors are agreed that the condition is most commonly found in middle-aged multiparous women.

In the case under discussion, the twisting of the pedicle evidently acted as the predisposing cause of the obstruction, the consequent swelling of the organ sufficing to obliterate the lumen of the bowel. The onset of acute gastric dilatation is interesting in view of the fact that the operation was conducted under spinal anæsthesia. The cause in this case appeared to be air-swallowing, although traction on the gastrosplenic omentum may have had some influence in its production.

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REFERENCES.

[1] Moynihan, Sir Berkeley, *Brit. Jour. Surg.*, 1921, viii, 307.
[2] Ledomski, *Khirurgia Mosk.*, 1908, xxiv, 559.
[3] Finkelstein, B. K., *Brit. Jour. Surg.*, 1914, ii, 68.

## HYDROCELE OF A FEMORAL HERNIAL SAC.

By HAMILTON BAILEY,

SURGEON TO THE DUDLEY ROAD HOSPITAL, BIRMINGHAM.

For fourteen years, L. M., age 58, had a right femoral hernia. About a year before she came under my observation she developed ascites due to cardiac failure, and the hernial sac became distended with ascitic fluid. After months of treatment with digitalis the general ascites completely abated, but the fluid in the femoral sac persisted. One week before admission to hospital

Fig. 127.

the swelling had been tapped. The distended femoral sac, as seen in the illustration (*Fig.* 127), is surmounted by a scab which formed around the site of this puncture, and the contents had become slightly infected. Three days later the sac, which by this time had become frankly purulent, was incised. After discharging for a fortnight, the wound healed, and the patient left hospital with no sign of the femoral hydrocele.

# *REVIEWS AND NOTICES OF BOOKS.*

**Scoliosis: Rotary Lateral Curvature of the Spine.** By SAMUEL KLEINBERG, M.D., F.A.C.S. Medium 8vo. Pp. 311 + xvi, with 140 illustrations. 1926. New York: Paul B. Hoeber Inc. London: Humphrey Milford. 30s. net.

WE may take it that this book embodies the practice followed at the New York Hospital for the Ruptured and Crippled, the work having been done in the clinic of Dr. Royal Whitman, to whom the author acknowledges his indebtedness in the preface. Looking at the book as a whole, we are impressed by the value of the letterpress and the care with which it has been written. It is evidently the result of much careful thought and painstaking work, and is not a mere collection of pictures held together by indifferent description.

Every aspect of the subject is dealt with, including treatment by operation. In describing the movements of the normal spine the author follows Lovett in dividing these into three only, viz., flexion, extension, and lateral flexion plus rotation. We cannot approve, however, his description of functional scoliosis, since he states on p. 38 that on bending forwards "one side of the back is higher than the other"—a statement which is inaccurate and misleading. It is generally admitted that on flexion a postural curve disappears, and there is no evidence of rotation towards the convex side. He makes no mention of the fact that the rotation, if present, in the lumbar region in such a case is on the *concave* side of the curve, is only due to inequality in the length of the legs, and is apparent and not real. Again, he states that when the child is asked to straighten up he can do so readily. In many cases this is certainly not true until after special instruction.

The vexed question of the importance of anatomical abnormalities at the lumbosacral junction as causes of scoliosis is well discussed, and the author, we think rightly, concludes that, though such abnormalities may "in a very small number" of cases account for the scoliosis, in most they are secondary to antecedent curvature of the spine. As usual, the reproduction of the radiograms is far inferior to that of the rest of the illustrations, and some are quite useless. It is pleasing to find the author insisting that symmetrical variations in number of the vertebræ have no influence on the causation of scoliosis. In the author's experience, scoliosis due to asymmetrical congenital abnormalities rarely passes beyond the mild and moderate in degree. We wish we could agree with him in thinking that paralytic scoliosis, if it cannot be corrected, "can surely be prevented from making progress." Tuberculosis is only mentioned with such relatively rare affections as osteomalacia, syphilis, spondylitis deformans, etc., and is dismissed in a line. No mention is made of the frequency with which lumbar caries produces a lateral as well as an anteroposterior curve.

In discussing the many theories as to the causation of the so-called static cases, which he apparently distributes into two groups—occupational and 'without evident causes'—he refers at length to Buchanan's work at the Hospital for the Ruptured and Crippled on affections of the vertebral epiphyseal plates as a possible cause: he makes no mention of Bankart's paper and the latter's application of Sherrington's work on postural tone to this subject. We agree with the author's outspoken condemnation of the value of the ingenious and complicated instruments for measuring the degree of deformity. He rightly says that when the improvement is so slight as to need mathematically accurate measurement to demonstrate it, it is not worth the effort expended to ascertain it. Again, we welcome his advice to err on the side of keeping a poliomyelitic child too long in the recumbent position rather

than allow him to sit up too soon. Hysteria is only mentioned as a possible cause, and nothing whatever is said as to the diagnosis of this extremely important, if rare, group of cases.

The chapters dealing with treatment are naturally the most important, and are, in fact, the most interesting. The author evidently believes in the possibility of at any rate improving the cases of structural scoliosis, and advocates vigorous methods of dealing with them. In all but the mildest cases he advocates forcible correction of the deformity, followed by the use of a brace or corset and gymnastic exercises. The chapter on the last is good, and states very clearly the object aimed at by exercises and the limitations of their use. We are glad to see the author insists on the necessity of the patient " constantly and consciously holding his body in the corrected attitude ". He evidently has little use for steel braces of any sort. On the vexed question of the value of passive stretching he decides that the method helps to reduce the deviation of the spine but has no effect on the rotation. The various methods of forcible correction are fully discussed. The Abbott flexion method, after a fair trial with the usual plaster jacket and with a steel brace designed by himself, he condemns as painful and distressing, and because it yields results no better than, if as good as, those achieved by other methods. The extension methods, with lateral traction, for the application of plaster jackets, the author uses extensively. Full details with illustrations of the way in which the author applies the jacket are given. With either method he claims improvement in 55 to 60 per cent of cases.

He is evidently enthusiastic on the results of the operative method of treatment, which, as used at the particular hospital at which he works, " bids fair to replace other methods of treatment in a large but selected group of cases ". The method advocated involves a stay of from three to four months in hospital. To begin with, the patient is placed on a convex frame for four to eight weeks, with traction on the head and pelvis. The operation includes portions of the Hibbs, Albee, and Forbes techniques. A long beef-bone graft is used in preference to one taken from the patient's tibia. Ten or twelve vertebræ are fused. After a week in bed the patient is again placed on the convex frame and traction reapplied. After two months a light plaster jacket is worn for six months, and then a corset, which is gradually discarded. As to results, in 31 out of 33 cases the improvement obtained by the preliminary traction has been maintained, and the author believes fixation and prevention of increase of the deformity has been achieved. Two cases relapsed. One patient died as a result of operation. The indication for operation is a progressive increase of the deformity, irrespective of the etiology of the scoliosis or the age of the patient. The way in which this subject is dealt with must prove of great use to the surgeon called upon to treat these cases. So impressed is the author with the improvement achieved by traction on a concave frame that he is disposed to use this method instead of plaster jackets in cases not requiring operation.

This book can be recommended to all seeking information on the subject. It should be read by every orthopædic surgeon.

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**The Inflammatory and Toxic Diseases of Bone: A Text-book for Senior Students.** By R. Lawford Knaggs, M.C. (Cantab.), F.R.C.S., Consulting Surgeon to Leeds General Infirmary, formerly Professor of Surgery in the University of Leeds. Demy 8vo. Pp. 416 + xii, illustrated by numerous photomicrographs by G. H. Rodman, M.D., Hon. F.R.P.S. 1926. Bristol: John Wright & Sons Ltd. 20s. net.

In his preface the author questions whether the title of this work accurately defines its scope. It is doubtful whether all the diseases of bone included within this volume are inflammatory or toxic; but there are also included descriptions of diseases in which the bone lesion forms by no means the chief part, of which the chapters on arthritis deformans and Charcot's joints are the most obvious examples. This remark is not intended to suggest any discontent with the author's selection, which is in most respects an advantage, because the author writes on what interests him and endeavours to correlate the different bone diseases with one another; in

fact the chief debt which teachers of surgery and students reading for the higher examinations will owe to him is the light he sheds upon the inter-relationship of osteitis deformans, osteitis fibrosa, and osteomalacia.

One began to read this book for the purpose of review, but soon found it so interesting and so sound a corrective to inaccurate or loose ideas on the pathology of the diseases of bone, that it has already served as a great help in the teaching of students. When one thinks of what had to serve in one's own student days as a description of that extremely important and common disease, acute osteomyelitis, and compares it with Mr. Knaggs' chapter, which no student could fail to understand, one is most envious of the surgical beginner who can gain his knowledge of this subject by the aid of so fascinating a mirror.

The illustrations are delightful; one reproduction after Howship of leontiasis ossea is perhaps the most beautiful thing to be discovered in any work of surgery.

To have produced this volume in the course of one's ordinary professional career would have been no mean achievement, but to have compiled so complete a pathological description of what most people regard as a relatively dry and uninteresting section of surgical literature after one has put aside ordinary professional duties is indeed a test of a very high endeavour and most conscientious sense of duty.

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**Clinical Surgical Diagnosis.** By F. DE QUERVAIN, Professor of Surgery and Director of the Surgical Clinic at the University of Berne. Translated by J. SNOWMAN, M.D. Fourth English edition. Medium 8vo. Pp. 937 + vi, with 750 illustrations and 7 plates. 1926. London: John Bale, Sons and Danielsson Ltd. 42s. net.

THIS new edition of de Quervain's *Clinical Surgical Diagnosis*, the last edition of which appeared in 1921, is a translation of the eighth German edition which was published in 1922, with the addition of a large amount of new material to be incorporated in a new edition shortly to be brought out in Germany. The principal alterations are in the chapters on the brain and the modern methods in diagnosis of renal function, while the chapter on the thyroid gland has been completely rewritten.

In dealing with his subject the author considers each part of the body in turn, and discusses the means of differentiating between the various pathological conditions which are to be met there. It is done in a very practical way: the most prominent symptom is taken as a starting-point and its probable cause considered, most attention being devoted to the more common diseases, whilst the rarer ones are treated in the proper perspective. The book is well written and easily readable. A conversational style is adopted as if a verbatim report of a series of clinical lectures had been recorded, and the stilted literary style which is so common among medical authors is avoided.

The only criticism we have to offer is when the author leaves his subject of diagnosis for that of pathology or treatment, but this is an infrequent happening. For example, on p. 140, when discussing sarcoma of the tonsil, we have: "Most of these sarcomata are of the lymphosarcomatous type and are not genuine tumours, even if they cause glandular metastases. Treatment by arsenic or X rays often causes them and their metastases to vanish, without leaving a trace." This may be the experience abroad, but it is certainly not the teaching or finding in this country. Again, the treatment of the peritonitis complicating acute appendicitis is discussed, and we have: "It must be washed out and thoroughly drained", a statement with the first part of which most of us strongly disagree.

The only noteworthy omission is that no reference is made to cholecystography after filling the gall-bladder with an opaque salt, a diagnostic method which has been sufficiently long in use, and is sufficiently useful, to warrant its inclusion in a book of this type. These are only minor points, and do little to detract from the value of the book as a whole.

The illustrations are particularly good; they are numerous and well produced, and it is almost possible to see a picture of every condition mentioned in the text, or at least of the ones more commonly met with. For a book of reference on the purely diagnostic side of surgery it still maintains its high position, and we can confidently recommend it to our readers.

**Manual of Operative Surgery.** By Sir HOLBURT J. WARING, M.S., F.R.C.S., Surgeon to and Joint Lecturer in Surgery at St. Bartholomew's Hospital, etc. Sixth edition. Crown 8vo. Pp. 868, with 618 illustrations. 1927. London: Humphrey Milford. 18s. net.

THIS well-known and widely-used manual hardly requires any detailed review. It first appeared in 1898, and from that date onward has maintained its character for clearness and simplicity. Like all small books it runs the risk of having its original character of brevity lost by constant additions which appear in each fresh edition. The present edition has been revised throughout and contains some very good additional illustrations by Maxwell. Probably still further improvement could be effected by the omission of operations rarely, if ever, performed, e.g., that for the suture of a badly lacerated spleen. The chapters on bones and joints have not undergone much change in the last ten years, and they represent the operative surgery class of work rather than a clinical presentation. All the figures of the suture of a ruptured patella show the sutures badly placed, with sharp angles and kinks. Formal excisions are described as the examinee requires to know them, but the modern operations of arthroplasty are not mentioned. We venture to suggest to the author that he ought to decide whether the book is primarily intended for the Final Fellowship examination or for the practising surgeon. At present it is too much for the former and not enough for the latter.

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**Thérapeutique chirurgicale.** By P. LECÈNE, Professeur à la Faculté de Médecine de Paris, and R. LERICHE, Professeur à la Faculté de Médecine de Strasbourg. Vol. I (pp. 644), General Principles, Limbs; Vol. II (pp. 508), Head, Neck, Thorax, Spine, Pelvis, Nose, Ear, and Throat. 1926. Paris: Masson & Cie. 10s. each volume.

VOLUME III, written by Lecène on the abdomen and genito-urinary organs, has already appeared. The three volumes complete a work for which the authors deserve the highest praise. Concentrating upon the subject of surgery as a therapeutic agent, it appears to us to be a publication of an original character. In each section the indications for surgical treatment are first dealt with. The various feasible surgical procedures are then described; and the results, immediate and remote, which may be expected of them are based both on the experience of the authors and on the statistics of other well-known surgeons. If the subject is one in which the author is specially interested, a most valuable résumé of his own choice of treatment for picked cases is appended. Every chapter is followed by a bibliography giving the more important references to the literature.

Since the work refers to surgical treatment, the problems of diagnosis are not referred to, except, as for example in dealing with certain breast tumours, when the first step in treatment may be establishing the nature of the tumour by operation. For the same reason pathology is not dealt with except in so far as a brief description of its pathology is necessary to indicate the particular condition under consideration. No instruction in pathology is given, but the familiarity with surgical pathology necessary for the proper understanding of the indications for treatment is assumed. The outlines, and not the details, of operative technique are described.

It will thus be seen that the scope of the work is narrowed down purely to the problems of the management of surgical cases; and if it be possible to write a book as a guide to 'surgical judgement' this must surely be one of the most worthy attempts ever made to accomplish so difficult a task. There are selected references to the best of all the European and American literature, and these in themselves, even apart from the carefully reasoned criticism of the authors, make the volumes worth reading. One feels that throughout the work everything has been carefully thought out in the light of ripe experience, and there is no suspicion of handing on advice with antiquity as its only justification. The familiarity of the authors with recent advances all over the world is as amazing as it is stimulating. The effects of the war are frequently to be noted.

In a work which is uniformly of such a high standard it is difficult to pick out the specially valuable sections; but those by Leriche on the general principles of

the treatment of wounds and on the blood-vessels, and Lecène's chapters on the skull and brain and on wounds and infections of the chest, are perhaps the most noteworthy. The part of Vol. II dealing with the ear, nose, and throat has been written by Professor F. Lemaître.

The authors explain that certain special subjects such as ophthalmic surgery and the orthopædic problems in children are not dealt with; but the principles laid down in these books deserve the careful consideration of all general surgeons—and physicians, for these volumes show repeatedly how modern advances are introducing fresh surgical procedures which may in time prove of value in diseases which up to the present have been considered 'medical', and in which surgery as a therapeutic agent has not been employed.

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**Cystoscopy.** By JAS. P. MACALPINE, F.R.C.S., Honorary Surgeon and Surgeon in Charge of the Genito-urinary Department, Salford Royal Hospital, Manchester. Large 8vo. Pp. 284 + xvi, with 181 illustrations and 12 coloured plates. 1927. Bristol: John Wright & Sons Ltd., 25s. net.

THE author states in his preface that, in the belief that there was a real need for a book on this subject, he has 'attempted the task'; after reading the volume very carefully and with intense pleasure, we venture to assure the author that his attempt has been extraordinarily successful, and that this book is a remarkable achievement; we consider it to be one of the best monographs we have ever read, and we predict that it will rapidly become the standard work on the subject.

We are extremely grateful to both author and publishers for the convenient form in which the work has been printed; at last one can study a book on cystoscopy without having to sit up to a table; it is beautifully printed, and there is hardly a printer's error in the whole work of 284 pages; the author's style is pleasant and easy to follow, and he has taken the utmost pains to make his meaning clear.

With a quite unnecessary modesty, the author apologizes for the limited number of cystoscopic drawings from which he had to choose; the twelve plates of coloured cystoscopic pictures are delightful, are beautifully reproduced, and have in no way suffered from their being presented on a smaller page than is usual in a cystoscopic atlas. There are also 181 other illustrations; of these, we can say that they are charmingly drawn, are entirely relevant to the text, and help materially in elucidating it.

The book is not merely a manual of cystoscopic technique, but contains a vast amount of clinical and pathological material; especially good are the sections on tumours of the bladder, on tuberculosis of the urinary tract, on ureteric catheterization, and on the diagnosis and treatment of stone in the ureter; a former article on symptomless hæmaturia is also included, and is well worth careful reading. One of the charms of the book is that, whilst Mr. Macalpine acknowledges the work of other urologists, one feels that he is relying so largely on his own experience.

Perhaps the two most striking chapters are those on the cystoscope and on the examination of a urological patient; we have read many accounts of the mechanism of the cystoscope, and have sometimes wondered whether the difficulties of understanding the somewhat complicated optical system of this instrument were not due to the fact that the writer himself was not altogether clear on the subject; no one could possibly think this of the present writer; the whole chapter is a model of lucid exposition, and we defy any person of average intelligence to read this section and not have a very reasonable idea of the mechanism of the cystoscope.

The sections on the care and sterilization of the cystoscope are full of valuable advice, and that on the tracing of faults in the electrical transmission should be studied by every surgeon who uses this instrument.

The following chapter, on the examination of a urological patient, contains a remarkably good account of how to use this instrument; the author is evidently a urologist of decided views, but he always gives ample reasons for the belief that is in him; we note that, after a prolonged trial of local anæsthesia, he has become an adherent of the sacral method, and we remark with sympathy his opinion that

" the best of all anæsthetics is a gentle and educated touch, and no other kind will make up for a lack of this ". We agree with him that the pain felt by the male during cystoscopy is due very largely to the straightening out of the more or less fixed curves of the proximal urethra, and that local anæsthesia does little to ameliorate this. He points out that the normal mucous membrane of the bladder is insensitive, and that painful sensations there depend on the presence of inflammation, and further, that the degree of pain is due much more to the depth to which the inflammation has progressed than to its superficial extent, as is well shown in tuberculous infection of that organ.

In this, as in all the other chapters, theory and practice are most skilfully interwoven, with the inevitable consequence that the reader is convinced that the author's methods of diagnosis and treatment are sound; his occasional references to the anatomy and physiology of the urinary tract add greatly to the enjoyment of the book.

After this pæan of praise, we hope the author will not take it amiss if we point out that there are some, though only a few, shortcomings in the volume; thus on page 156 the word 'faceted' would look more at home with another *t*, and on page 105 the word ' serpigenous ' should surely be serpiginous: again, on page 76, the name 'trigonitis pseudo-membranosum' gives a shock to the most amateur of classical scholars; we have looked up Pilcher's *Practical Cystoscopy* (2nd edition, 1915, p. 222), and there we find a condition of the bladder described as 'œdema trigoni membranosum'; can this be the same?

It is only our personal opinion, but we think that, in future editions, the section on malakoplakia might well be omitted; the information given on this rare condition is nearly all within inverted commas, and one would so much prefer to have the author's personal experience on some subject with which he is well acquainted. If one might make a suggestion for the future, the question of vesical fistulæ would well deserve a separate chapter; also the writer's experience of the after-effects of implanting the divided ureter into the bladder; it has been stated lately by some French urologists that the corresponding kidney after such an operation becomes eventually functionless; Mr. Macalpine must have seen many such cases, and his observations would be highly interesting and valuable; moreover, the question is entirely relevant to such a book, as it can be settled only by skilled cystoscopy.

There is a good index, but no bibliography; there are a few references in the text, but we think future editions would benefit by a short chapter devoted to a discussion of those works on urology of which the author has proved the value; the usual list of works appended at the end of a book is of little value, as there is no discrimination as to their relative merits, and the average surgeon has not the time or the inclination to look them up.

In conclusion, we congratulate Mr. Macalpine on this book; we think he is *facile princeps* in the art of writing about cystoscopy, and, if it is true that easy writing makes very hard reading, we fancy that he must be one of those with an infinite capacity for taking pains.

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**Le Cancer de l'Œsophage.** By L. Bérard, Professeur à la Faculté de Médecine de Lyon, and A. Sargnon. Medium 8vo. Pp. 450, with 76 illustrations. 1927. Paris: Gaston Doin & Cie. 77 fr.

This volume is one of a series which includes monographs on cancer of the intestine, kidney, rectum, thyroid, uterus, eye, and upper air-passages. It is a monument of industry, a most extensive search having been made in the world's literature for the information the book contains. It may be said to summarize almost everything that is known with regard to cancer of the œsophagus. Its faults are that it is very long, it is inclined to be rambling in many parts, and it does not point the way clearly to any definite conclusions. The length is due to unnecessary repetition, and reference to subjects other than cancer. It is indefinite in that the opinions or results of many observers are recorded, and the reader is often left to form his own conclusions from this rather confusing mass of statistics.

The book starts with a valuable anatomical section which contains a well-illustrated account of the problem of surgical approach in operations on the œsophagus. The chapters on etiology and pathology with their bibliography make it a useful book of reference to anyone working on this subject. The sections on diagnosis and treatment contain accounts of all the useful modern methods, but do not exclude those that are obsolete, and thus make tedious reading. They do not contain any 'original' matter, and they offer no hope of a better outlook as regards the curative value of operative treatment.

The book is illustrated by numerous drawings which on the whole are reasonably clear.

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**Baillière's Synthetic Anatomy: A series of Drawings on Transparent Sheets for facilitating the Reconstruction of Mental Pictures of the Human Body.** By J. E. CHEESMAN, Deputy Medical Officer of Health for Leyton, London. Size of Plates 7½ in. × 9 in. Part I. The Upper Arm and Shoulder; Part II. The Forearm; Part III. The Hand. To be completed in 12 Parts. 1926. London: Baillière, Tindall & Cox. 2s. 6d. each net.

IT is undoubtedly true that the average student of anatomy, when apart from actual dissection, often finds a real difficulty in forming a mental picture of the body structure in relationship to planes. Dr. J. E. Cheesman, in his *Synthetic Anatomy*, has produced what at the present time is perhaps the best and most convenient substitute for the actual body. The idea is not entirely original, but we must congratulate the author on his novel conception of superimposing one plane of tissue drawings upon the other on transparent sheets. The picture thus presented permits the student to carry his eye from one body plane to another without having to disturb his chain of thought by removing one layer from a model to expose the next, or by folding back the leaves of a book.

Three parts of the twelve which constitute the atlas have been published. These deal with the upper extremity. Each part consists of some twelve coloured representations, and each of these is carefully calculated to suit the progress of the reader's thoughts. In anatomical details the work is extremely accurate. Drawings and colourings are not lacking in artistic finish. We feel certain that this work will form a great asset to students in their study of anatomy, and to all for purposes of rapid revision. Each of the twelve parts may be purchased separately at the reasonable price of half a crown. We congratulate the author on the novelty of his conception and have every confidence in recommending his production.

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**Anatomie élémentaire des Centres nerveux et du Sympathique chez l'Homme: Vie de Rélation et Vie végétative.** By P. GILIS, Professeur d'Anatomie à la Faculté de Médecine de Montpellier. Crown 8vo. Pp. 232, with 35 illustrations. 1927. Paris: Masson et Cie. 20 fr.

THIS little book, written by a professor of anatomy, is intended to give students a comprehensive survey of the anatomy of the central nervous system, and also of the sympathetic nervous system. It is intended also to bring out that these are not two separate systems but one system, forming one unit, each part of which reacts constantly on the other. The author holds that it is necessary to teach students this truth now that the disorders of the central and of the sympathetic nervous system play such an important part in medicine and surgery. It is a useful little book, written on sound educative lines.

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**Hernia and Hernioplasty.** By ERNEST M. COWELL, D.S.O., M.D., F.R.C.S., Surgeon, Croydon General Hospital; Hunterian Professor R.C.S. Demy 8vo. Pp. 128 + xvi, with 64 illustrations and 8 plates. 1927. London: H. K. Lewis & Co. Ltd. 9s. net.

THERE is no finality in medicine or surgery. There is more to be said even on the subject of hernia and its radical cure. The older surgeons of to-day have been inclined to think that the operation for the cure of inguinal hernia is of the 'sealed pattern' variety. Good as this operation has proved to be, it still fails in a small

percentage of cases, and this little book shows how this small number may be still further reduced by employing some of the plastic methods recently introduced.

It is a really useful book and well repays perusal. If a friendly criticism is allowed, and the book goes to a second edition, we would urge that the illustrations be improved. The photographs are not sufficiently clear, and semi-diagrammatic drawings would be more useful. Then there is a curious little mistake the author makes in stating that "the fold of the groin indicates the position of Poupart's ligament". With the exception of these minor points we can strongly recommend the book.

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**Diagnostische und therapeutische Irrtümer und deren Verhütung.** Edited by Professor SCHWALBE. Eighth part: Peritoneum (KLEINSCHMIDT), Appendicitis (PAYR), External Hernia (HOHLBAUM). Pp. 261, with 36 illustrations. 1926. Leipzig: Georg Thieme. M. 16.50.

THIS is a part of a large System dealing with diagnosis and treatment. It is a very careful and complete discussion of the mistakes that are liable to be made, relating both to the main diseases under consideration and also all the possible complications. The treatment of each disease and complication is also dealt with very fully, special attention being paid to the things which may go wrong at every turn.

There is perhaps a little overlapping in the first two sections of the book, because the first deals with peritonitis including appendicitis, and the second is concerned only with appendicitis in its various forms and with all possible complications. This section (132 pages) by Professor Payr, of Leipzig, represents the bulk and the most important part of the book, and constitutes a most valuable monograph on the subject. The chief drawback to these articles is perhaps the absence of any tabulation, so that any particular point on which it is desired to seek information has to be searched for rather closely; but this drawback is minimized by an excellent index. The illustrations are scanty, and this fact also makes the reading of the text more tedious; but as a thorough discussion of the difficulties in diagnosis and treatment of peritonitis, appendicitis, and external hernia, it forms a most valuable work of reference.

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**Les Pancréatites aigües chirurgicales.** By PIERRE BROCQ, Chirurgien des Hôpitaux. Pp. 188, with illustrations. 1926. Paris: Masson et Cie. 4s. 2d. net.

IT is always instructive to reconsider a series of observations made over a period of years and to rearrange facts in their proper perspective. The author has previously made twelve contributions to the literature on the subject of acute pancreatitis, and in his latest book reviews his published cases, together with twenty new ones. The book is based on a study of 340 cases of the various types of acute pancreatitis, together with a large number of cases of the disease produced experimentally in dogs. He is, therefore, well qualified to discuss the subject and to draw conclusions as to the clinical and pathological course of the disease.

On the clinical side the picture of the disease is clearly drawn, and its progress from the early vague attacks of abdominal pain to the acute attack, its operative treatment and sequelæ, are laid down in detail. The only criticism which we would offer is that he is a little too insistent that a cholecystotomy should not be performed when operating for the acute disease.

On the pathological side the picture drawn is logical and convincing. It is maintained that tryptic digestion and not infection is the starting-point, and that if the latter occurs it is secondary and accidental. He says that the activation of the trypsinogen is usually due to something gaining access to the pancreas along the duct of Wirsung, and, if he is perhaps a little too prejudiced as to this explanation, he brings much evidence, both clinical and experimental, to support his views.

This book fulfils a very useful purpose in putting clearly the present position of acute pancreatitis in all its aspects. The text is good, the illustrations are well reproduced, and we can confidently recommend it to those who are interested in the subject.

**Practical Methods in the Diagnosis and Treatment of Venereal Diseases.** By DAVID LEES, D.S.O., M.A., M.B., F.R.C.S. (Ed.), Surgeon in Charge of Venereal Diseases, Royal Infirmary, Edinburgh. With an Introduction by WM. ROBERTSON, M.D., F.R.C.P., D.P.H., M.O.H. Edinburgh. Crown 8vo. Pp. 605 + xvi, illustrated. 1927. Edinburgh: E. & S. Livingstone. 15s. net.

IF a popular novelist could be induced to rewrite this book it might become a 'best seller' in the medical world. Unfortunately the literary standard falls short of the clinical, and the numerous split infinitives and pleonasms, coupled with the inordinately frequent use of such words as 'carefully', 'exhibition', 'exhibited', tend to jar on the reader.

The section dealing with syphilis is extremely good and the illustrations are excellent; differential diagnosis, which is so important, is well done. That on gonorrhœa is hardly so good, but the teaching is eminently sound.

Vaccines are accorded the importance they deserve, but diathermy is dismissed in two lines, and hexyl-resorcinol is not even mentioned. Such expressions as 'normal' (for physiological) saline, 'male gonorrhœa', 'the venereal patient', and 'alternately' (for alternatively) should not occur. The general impression conveyed is that the materials have been carefully and laboriously collected and then written up hurriedly and without due thought. A second and improved edition may be predicted in the near future.

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**Guy's Hospital Reports.** Edited by ARTHUR F. HURST, M.D. Vol. LXXVII (Vol. VII, Fourth Series), No. 1, January, 1927. Medium 8vo. Pp. 126, illustrated. 1927. London: *Lancet* Ltd. Annual subscription, £2 2s. net; or 12s. 6d. net per issue.

THIS volume does not contain much of interest to surgeons, beyond an article by W. H. Ogilvie on "Two Cases of Epitheliomata Occurring Simultaneously in Different Parts of the Body". These occurrences are rare, but are always interesting and justify publication.

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**LISTERIANA.**

1. **Reminiscences of Lister, and of his Work in the Wards of the Glasgow Royal Infirmary.** By Sir HECTOR CLARE CAMERON, C.B.E., LL.D., M.D., etc., Consulting Surgeon, Glasgow Royal Infirmary. Medium 8vo. Pp. 45, with 3 plates. 1927. Glasgow: Jackson, Wylie & Co. 2s.

2. **Lister as I Knew Him.** By J. R. LEESON, M.D., C.M. Ed., F.L.S., F.R.A.S., Senior Consulting Physician, St. John's Hospital, Twickenham. Demy 8vo. Pp. 212 + xi, with 2 plates. 1927. London: Baillière, Tindall & Cox. 8s. 6d. net.

3. **Lister and the Lister Ward in the Royal Infirmary of Glasgow.** Royal 8vo. Pp. 132 + xvi, with several plates. 1927. Glasgow: Jackson, Wylie & Co. 12s. 6d. net.

THESE three volumes are the direct outcome of the Lister Centenary. Two of them can be read with pleasure; the third only with shame not unmingled with righteous anger.

Sir Hector Cameron and Dr. Leeson are both hero worshippers. Their reminiscences of Lister whilst he taught and worked at Glasgow are full of interest, and form a fitting supplement to Sir Rickman Godlee's *Lord Lister*. They tell of him as a man with like parts as ourselves, but saved by his Quaker ancestry from similar passions. Both speak of the personal affection he inspired; of his minute attention to detail; of the unswerving pursuit of his ideal; of his professional isolation; of his entire absence of self-seeking; of his generous nature; of his kindliness of heart; and of his happy, though childless, home life. Working with him and under him they seem to have been ignorant of his true greatness or of the revolution which he was effecting in the practice of surgery. They recognized that to him each patient was a poor man or a poor woman whose feelings were to be respected and whose wounds were to be healed more quickly and more safely than those of his

colleagues. They saw a constant succession of surgeons from foreign countries who followed his practice, but of the great underlying principles which he was slowly formulating they remained in ignorance. Dr. Leeson's book is especially valuable because he went straight from the newly-built St. Thomas's Hospital in London, where Miss Nightingale had already introduced the modern nursing system, to the Royal Infirmary at Glasgow, which maintained the old tradition of ancient wards and inferior nursing. In London he left hospitalism in spite of the palatial housing; in Glasgow he found healing by first intention in the most inferior surroundings. How this occurred Dr. Leeson explains most clearly.

*Lister and the Lister Ward* is a record of the most astounding piece of wanton vandalism which has occurred for many years past. It is of a piece with the destruction of the manuscripts of John Hunter by Sir Everard Home. The Royal Infirmary of Glasgow has recently been reconstructed, and the opportunity occurred of keeping the male accident ward in which Lister did his monumental work. The Managers at first intended to preserve it, but evil counsels afterwards prevailed; the vote was rescinded, and, in spite of remonstrances received from many parts of the world, the ward was pulled down and the site is now derelict. The Wellcome Historical Medical Museum, thanks to the energy of Mr. C. J. S. Thompson, then the Curator, secured a part of the ward and some of its furniture. These have been put together, and it is possible to get an idea of what the ward looked like; but the genius loci is wanting, and what should have been preserved for ever has now been permanently destroyed.

This volume records, so far as can be done in words, plans, and illustrations, the history and appearance of the Infirmary buildings. It is thus a fitting supplement to Sir Hector Cameron's *Reminiscences* and to Dr. Leeson's account not only of Lister as a man but of the conditions under which he worked first in Scotland and afterwards in England. The three books ought to be read by every operating surgeon, for they will teach him how great an advance has been made by a single generation.

**Lister's Centenary Exhibition at the Wellcome Historical Medical Museum: Handbook.** Pp. 216, illustrated. 1927. London: The Wellcome Foundation Ltd.

**The Wellcome Historical Medical Museum: Handbook indicating the chief Features and Objects Exhibited in the Museum.** By HENRY S. WELLCOME, Director, and L. W. G. MALCOLM, M.Sc. (Cantab.), F.R.S.E., Conservator. Pp. 118, illustrated. 1927. London: The Wellcome Foundation Ltd.

MEMBERS of the medical profession throughout the world who are interested in the history of their calling owe a debt of gratitude to Mr. Henry S. Wellcome for the magnificent museum he has gathered together in London with the help first of Mr. C. J. S. Thompson, more recently of Mr. L. W. G. Malcolm. The Lister Centenary Exhibition owes much to both these Conservators. The energy and foresight of Mr. Thompson secured a part of the Lister Surgical Ward and of its furniture when the Directors of the Royal Infirmary at Glasgow pulled it down. The organizing power of Mr. Malcolm has led to a rare display of Lister relics which are catalogued in the two volumes just issued by the Wellcome Foundation Limited. Sir Hector Cameron introduces them with a lively account of the evolution of Lister's system of antiseptic surgery. He tells his story with the advantage of first-hand knowledge. He served for two periods as Lister's house surgeon, was Professor of Clinical Surgery in the University of Glasgow, and remained a friend of the Master throughout his life. Sir Hector's introduction is followed by a Life of Lord Lister, and a very useful list of his house surgeons and dressers in Glasgow, Edinburgh, and London. Both books are remarkably well illustrated.